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IN THIS ISSUE

Anesthetic Accidents
Harold Carron

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Guy A. Caldwell

Malformations of Thoracic Aorta
Jesse E. Edwards and H. Milton Rogers

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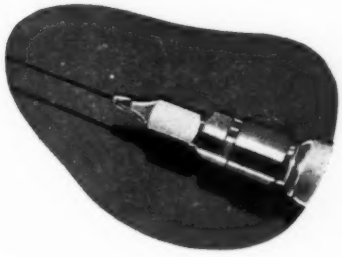

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Prevention and Treatment of Anesthetic Accidents

HAROLD CARRON, M.D.
TAMPA

Anesthetic accidents and complications are the major hazards in surgery today. Few fatalities occur as a direct result of surgical trauma, but a carelessly administered anesthetic can turn a surgical triumph into a veritable catastrophe. Most anesthetic accidents occur as a consequence of poor patient preparation, improper premedication, poor choice of anesthetic agent or technic, or careless administration of the anesthetic. For the sake of convenience, one may classify anesthetic accidents according to the sites of insult, remembering, however, that often the fatal complication will occur as a result of trauma at a distant site. The main groups encompassing these anesthetic difficulties are in order of their frequency of occurrence (1) respiratory, (2) circulatory, (3) cerebral, and (4) peripheral. In addition, there are those accidents occurring while the anesthetic is being given but not necessarily related to its administration.

Respiratory Accidents

In the first group, the respiratory accidents, there are those related to poor patient preparation. This situation most frequently exists in acute intra-abdominal catastrophies such as a ruptured viscus or acute intestinal obstruction. In these emergency cases, the patients are in a state of impending shock. There is fluid imbalance with a loss of electrolytes and fluid into the intestine or peritoneal cavity. Upon the induction of anesthesia, there occurs a relaxation of the pharyngeal and gastric sphincters with a consequent reflux regurgitation of gastric and intestinal contents into the pharynx. Aspiration of vomitus and laryngospasm occur with subsequent frequently fatal results. Abdominal distention of any degree interferes with adequate respirations, particularly if the patient is placed in the Trendelenburg position. Gastric or intestinal suction should be employed in all such cases, and intravenous fluids, plasma, or blood should be administered until the pulse rate falls below 100 and the blood pressure reaches a stable level. Narcotics administered to patients in shock or impending shock should be given intravenously, since a deficient circulation will not pick up hypodermic medication until the shock is corrected. Only after these conditions have been satisfied is it safe to proceed with the administration of an anesthetic. It is never safe, regardless of the desirability of the obtained relaxation, to administer a spinal anesthetic to a patient with either intestinal obstruction or a ruptured viscus, for it is the sympathetic system alone in these patients that is maintaining the circulation. Administration of a spinal anesthetic removes that last constrictor influence on the peripheral vascular system, and irreversible shock usually results.

Proper premedication of the surgical patient includes the use of a narcotic to decrease metabolic activity, provide sedation, and decrease the amount of anesthetic required. A belladonna derivative, preferably scopolamine over atropine, should be given to obtund vagal reflexes, provide psychic sedation, decrease secretions, and furnish amnesia for the visit to the operating theater. Preferably, these drugs are given one to one and one-half hours prior to the time of the scheduled operation, for the narcotics reach their peak of efficiency in that time when administered hypodermically. If time does not permit of this interval, the premedication should be given intravenously, following which maximum effect will be

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obtained in 10 minutes. Premedication given "on call to the operating room," or while the patient is on the stretcher on the way to the operating room frequently results in excessive salivation with pulmonary aspiration during anesthetic induction. Induction of anesthesia is delayed by struggling of the unsedated patient. Apnea frequently results when the maximum effect of the anesthetic coincides with the maximum depressant effect of the premedication in the middle or at the termination of the operative procedure. Hiccups may result, particularly during induction with the intravenous barbiturates, if the vagal reflexes are not obtunded by the use of belladonna derivatives preoperatively.

By far the greatest number of anesthetic difficulties occur during the administration of the anesthetic and particularly during the induction period. How frequently have you heard that dreaded laryngeal stridor of laryngospasm occurring with an intravenous barbiturate induction, looking down to see a cyanotic patient making violent respiratory efforts but moving no air? Laryngospasm occurs most frequently when there is pharyngeal or laryngeal stimulation due to an irritant concentration of anesthetic gases or vapors or too early introduction of an airway. It may occur with premature stimulation of the patient after the intravenous administration of the barbiturates. Laryngospasm will invariably occur with the use of the intravenous barbiturates when there is dilatation of the anus, dilatation of the vagina, periosteal stripping of a rib, or any manipulation about the neck. Intravenous barbiturates should never be used for anesthesia about the head, neck, nose or throat for fear of fatal laryngospasm unless there is an endotracheal tube in place. It is far safer and much easier on both the patient's and surgeon's coronaries to use ether insufflation anesthesia for head, neck, and oral surgery when the facilities for endotracheal intubation are not at hand.

When laryngospasm does occur, its first treatment consists of positive pressure oxygen with an anesthetic bag and mask. Intermittent positive pressure on the rebreathing bag with oxygen alone will break up the laryngospasm in most cases. When this method fails, the intravenous use of curariform drugs is recommended. When it then becomes possible to force oxygen through the spasming larynx, intermittent positive pressure should be continued until the curariform drug is metabolized. If all such measures fail, and definitely before the circulation begins to fail, an endotracheal tube should be inserted, or a tracheotomy should be performed.

Pulmonary edema occurring during the period of anesthesia is usually the result of the patient breathing against obstruction of the airway. Noisy respiration, while it informs the anesthetist of continued respiratory effort on the part of the patient, is of necessity obstructed respiration. Obstructed respiration leads to inadequate respiratory exchange with its attendant deficient oxygenation, increased carbon dioxide retention, and subsequent development of pulmonary edema. The anesthetized patient has a labile vascular system, reacting unfavorably to changes in position and particularly to the overadministration of intravenous fluids. Pulmonary edema may result from overloading of the right side of the heart with intravenous fluids, and their administration should be judicious even in a healthy patient. When given intravenously to the average patient, 500 cc. per hour can be handled without difficulty. The hypertensive patient or the one with borderline compensation can handle much less. When pulmonary edema occurs, all intravenous fluids should be stopped, the patient should be placed in the reverse Trendelenburg position, tourniquets should be applied to the extremities, and oxygen should be administered under positive pressure. Hypoxia developing as a result of pulmonary edema increases the stimulus for further pulmonary secretions, which reflex may be inhibited by oxygen under positive pressure. Intravenous aminophylline may be of some value, but atropine should not be used for, by increasing the heart rate, it decreases cardiac output.

Laryngeal injuries result from traumatic endotracheal intubation, either through the use of too large a tube, or from attempt at intubation under too light anesthesia. Most laryngeal injuries heal spontaneously, but when they occur in young children, they may result in fatal laryngeal edema. When laryngeal edema occurs or when it might be expected to occur in children, the use of an aerosol oxgyen tent with a detergent added to the vapor may prove lifesaving. Tracheotomy equipment should be immediately available and should be used on the first indication of persistent respiratory difficulty.

Liquid ether pneumonitis is mentioned only as a precaution against its occurrence. Overfilling of the ether vaporizer and sudden release of a kinked tubing from the ether machine are the

commonest causes of this accident. Ether pneumonitis is a definite phlegmon of the pulmonary epithelium and requires prolonged positive pressure oxygen and supportive and antibiotic therapy.

During the administration of an anesthetic, coughing and laryngospasm may occur with sudden increases of anesthetic concentration and diaphragmatic or periosteal stimulation under light anesthesia. Respiratory arrest occurs with an overdose of the anesthetic agent or with massive doses of curariform drugs. The occurrence of the cessation of respiration in itself should cause no alarm, but treatment should be immediate. The anesthetic should be withdrawn and artificial respiration should be applied by intermittent compression of the rebreathing bag of the anesthetic machine. Manual compression of the patient's thoracic cage is heroic treatment but unnecessary when an anesthetic machine and oxygen are available.

Atelectasis results from a combination of excessive secretions trapped in a major bronchus by depressed respirations. This condition is most apt to occur in surgery of the upper part of the abdomen, where there is continued and prolonged retraction against the diaphragm with consequent compression of the lower lobes of the lungs. Its frequency of occurrence may be decreased by the careful use of muscle-relaxant drugs during surgery, by the judicious use of sedatives, and by encouraging frequent coughing postoperatively.

Circulatory Accidents

Hypotension occurring during a surgical procedure on the anesthetized patient usually falls into two classifications. The first of these, primary or neurogenic shock, occurs with prolonged stimulation under light anesthesia, as a result of prolonged abnormal operative positions, or as a result of vagal or traction reflexes. It is characterized by a fall in systolic pressure of considerable degree with only a slight fall in diastolic pressure and little change in pulse rate. The condition is due to a peripheral vasodilatation, is usually transient, and responds rapidly to the use of any of the vasopressor drugs. Secondary or surgical shock results from excessive traumatic manipulation, blood or fluid loss, or peritoneal contamination. This type of shock is characterized by a decided fall in both systolic and diastolic pressures with a definite increase in pulse rate, profuse cold perspiration, peripheral cyanosis, and sighing respirations. Physiologically there is present a high degree of peripheral vasoconstriction, and consequently the use of vasopressor or constrictor drugs is of no value. Treatment consists of the use of plasma volume expanders such as dextran, plasma, or blood until the pulse and blood pressure reach preshock levels.

Hypertension occurs under anesthesia usually as a result of carbon dioxide retention due to soda-lime exhaustion, respiratory obstruction, injudicious use of muscle-relaxant drugs, or the overuse of pressor amines. Hypertension resulting from such exogenous sources results in increased bleeding and hyperventilation with exaggerated abdominal movements, and may produce a rupture of sclerotic blood vessels.

Arrhythmias occurring under anesthesia, be they extrasystoles, auricular fibrillation, or ventricular fibrillation, are cause for concern. In the presence of adequate oxygenation and adequate elimination of carbon dioxide, arrhythmias rarely occur. The use of epinephrine, particularly when cyclopropane is being administered, may result in ventricular fibrillation. The treatment of the lesser arrhythmias consists of discontinuing the anesthetic and assisting the normal respiratory movements by manual compression of the rebreathing bag. The gaseous or vapor anesthetic should be frequently emptied from the anesthetic bag during this process. Ventricular fibrillation may be diagnosed by a sudden absence of pulse and blood pressure occurring between two beats with cessation of respiration, and it requires heroic measures. The left thoracic cage should be opened immediately without aseptic preparation in the fourth interspace, and the heart should be massaged vigorously and at a rate of 30 to 40 times per minute. At the same time artificial respiration with oxygen should be maintained, preferably through an endotracheal tube or with a bag and mask if a tube is not available. A mixture of 9 cc. of 1 per cent procaine solution and 1 cc. of 1:1000 epinephrine solution is suggested by some for injection into the left ventricle. A defibrillator should be used if available. Massage should be continued until normal rhythm is restored or until all cardiac activity ceases. The sooner cardiac massage is started following the onset of ventricular fibrillation, the greater the likelihood of the re-establishment of a normal rhythm. There is no time for less heroic measures, consultations, or procrastination.

Cardiac arrest, occurring usually from oxygen lack to the myocardium from any cause, requires the same immediate treatment as ventricular

fibrillation. The atonic heart, however, will not respond to the use of a defibrillator, but continuous and vigorous massage will maintain the circulation.

Cerebral Accidents

Cerebral complications of anesthesia are invariably the result of anesthetic mismanagement somewhere else along the physiologic tree. Cerebral thrombosis may result from a prolonged period of untreated hypotension, and hemorrhage may occur as a result of a hypertensive episode during anesthetic induction or maintenance. Anoxia and prolonged hypoxia, however, are the most frequent of the cerebral complications. Even a transient period of cerebral anoxia or prolonged hypoxia will produce irreversible damage to the nervous system and primarily to the frontal lobes. All cerebral oxygen lack results in the development of cerebral edema with signs indicative of increased intracranial pressure such as vomiting, reflex changes, respiratory irregularities, coma, convulsions, and even death. The treatment of cerebral anoxia, when it occurs, must be undertaken without delay to prevent permanent damage to the brain. The patient's head should be elevated, convulsions should be controlled with intermittent administration of ether, the resultant hyperthermia should be treated with alcohol sponges and antipyretics, and human serum albumin should be given intravenously in an attempt to produce cerebral dehydration. If unilateral convulsions occur, the contralateral stellate ganglion should be blocked in an endeavor to increase the cerebral circulation. Oxygen by tent or nasal catheter is a necessity.

Convulsions occurring under anesthesia are usually due to retention of carbon dioxide, but may occur as a result of toxemias, overdosage of atropine or vinethene, and dehydration hyperthermia in children. Their treatment consists of adequate oxygenation, intravenous fluids, short-acting barbiturates, and correction of the causative factor.

Peripheral Accidents

Among the peripheral injuries or accidents that may occur under anesthesia, one of the commonest is the development of gastric dilatation. This condition is most prone to occur in the patient with an obstructed airway with resultant "air sucking" due to increased respiratory effort. Gastric dilatation should be suspected postoperatively in every case in which there are a rapid pulse, a fall in blood pressure, and distress in the upper part of the abdomen when the other signs that might suggest shock are not present. Treatment consists in the insertion of a Levin tube with gastric suction for a few hours. Fall in pulse rate and elevation of blood pressure occur almost immediately following gastric intubation.

The commonest nerve palsies occurring under anesthesia are those involving the ulnar and peroneal nerves. The ulnar nerve is particularly vulnerable in the olecranon fossa because of pressure on the side of the operating table. Two to six weeks are required for return of function of this nerve following injury. The peroneal nerve may be injured when patients are maintained in stirrups over a protracted period. It is now customary in most institutions to pad knee crutches and stirrups with a 1/2 inch thickness of foam rubber to prevent injury of the peroneal nerve and also thrombophlebitis due to pressure on the popliteal vessels.

Back injuries may result from improper positioning on the operating table and most commonly occur when the patient's legs are placed in or taken out of the stirrups one at a time. Both legs should be raised and lowered together to prevent undue strain on the sacroiliac and lumbosacral joints.

Eye burns and injuries may occur from liquid ether during open drop anesthesia or from irritation of the anesthetic gas in inhalation anesthesia. The eyes of elderly patients, infants, and patients with Graves' disease are particularly susceptible. A small amount of any type of ophthalmic ointment placed in the patient's eyes after induction of anesthesia will prevent conjunctival irritation and corneal ulceration. Sloughs at injection sites result from the intravenous injection of irritant drugs or the extravascular injection of the intravenous barbiturates. When they occur, phlebitis and periphlebitis are best treated by the application of moist heat and elevation, and severe cases may require debridement or sympathetic blocks to relieve the pain and swelling.

The commonest complication of spinal anesthesia is postspinal headache. Its incidence may be minimized by the use of small caliber spinal needles and avoidance of trauma to the dura. When such a headache occurs, the use of the supine position, epidural saline, caffeine and analgesics may be of some benefit. The neurologic sequelae of spinal anesthesia, namely, nerve root neuralgias, cauda equina syndrome, and paralyses,

are best avoided through the careful introduction of the spinal needle and the avoidance of too concentrated solutions of anesthetic drugs. If the patient complains of pain in either extremity or in the perineum at the time the needle pierces the dura, the needle should be withdrawn and reinserted, for it is undoubtedly within a nerve root sheath and injection of the drug in this location may produce permanent neurologic damage.

Tracheal collapse may occur during the course of a thyroidectomy when adenomatous tumors have caused erosion of one or more tracheal rings. Diagnosis is made by observation on the development of tracheal obstruction during the course of the surgery. Attachment of the strap muscles to the collapsed trachea may suffice to correct the condition, but tracheotomy may become necessary. Pulmonary embolism and coronary thrombosis may occur during the course of the most skillful administration of an anesthetic, and treatment is the same whether the patient is anesthesized or not. Air embolism is an uncommon condition that may occur with laceration of the jugular vein, and diagnosis may be confirmed, when the condition is suspected, by the auscultation of air within the heart chambers. Treatment consists of immediate Trendelenburg position, vasopressor drugs and plasma intravenously, and oxygen with or without artificial respiration as required. Thrombophlebitis, while its occurrence is usually during the postoperative period, frequently has its groundwork laid, as mentioned previously, in improper positioning on the operating table with pressure over main venous trunks. Treatment consists of the use of adrenergic blocking agents, elevation of the involved extremity, anticoagulants and sympathetic nerve blocks.

Transfusion reactions under anesthesia are often masked by the anesthetic, but should be suspected in any case in which cyanosis, urticaria, or abrupt changes in pulse and blood pressure develop within a short time of the onset of administration of blood. If it is determined that the patient has received incompatible blood, the patient should be alkalinized immediately with intravenous molar lactate or sodium bicarbonate to prevent renal failure, and compatible blood should be administered to replace that destroyed by hemolysis.

Conclusion

Safety is the watchword in the administration of any anesthetic. Careful patient preparation and premedication will prevent many anesthetic accidents. How dangerous is that request for "just a whiff of gas" for the 300 pound patient; or, "give the patient just a little pentothal for this case won't take long." While some surgery may be considered minor, there is no such thing as a minor anesthetic. From the moment the mask is placed over the face of a patient or the needle inserted into a vein, that patient's life is in the hands of the anesthetist at the head of the table. The incidence of difficulty during anesthesia will be appreciably decreased if the anesthetist is considered part of the surgical team and is permitted to administer the anesthetic he considers most suitable, and more important, with which he is most familiar.

420 West Lafayette Street.

Discussion

Dr. Ralph S. Sappenfield, Miami: I am happy that an anesthesiologist, especially Dr. Carron, has had the opportunity to speak to this meeting. I appreciate his careful and detailed organization of such a big subject. Time seems to be a factor that keeps coming up when we think of accidents and complications occurring with surgery. I should like to emphasize Dr. Carron's point that we always have time to administer premedication since it can be given intravenously and also that we now have bellafoline® to add to the premedicant. Bellafoline® dries as well as scopolamine, but like atropine provides no psychic sedation but will obtund vagal reflexes more than either. There is a mutual problem of the surgeon and the anesthesiologist to check the patient preoperatively, to have the blood volume, the red blood cell count and/or the hematocrit reading within normal range. The failure to do so accounts for increased morbidity and mortality postoperatively.

Secondly, time comes into play when the surgeon, not having seen his patient in the hospital, wishes to speak to him before he is put to sleep. What is said the patient does not remember if he is properly premedicated. The surgeon then goes into the scrub room to use one of the new scrub soaps such as pHisoHex® or Septasol,® returning to the operating room after a three to five minute scrub. During this short time he expects the patient to be anesthetized, to be positioned, to be prepared, to be draped, and all waiting his entrance. This expectation tends to cause the anesthetist or anesthesiologist literally to slug the patient to sleep in order to accomplish these objectives. This tendency definitely increases the percentage of complications.

Thirdly, time is important throughout the procedure, and it should be conserved on every turn. Even with good anesthesia, increased time increases complications.

Dr. Carron spoke only briefly of spinal anesthesia in the presence of intestinal obstruction. May I remind you of the resultant constriction of the intestine by the circular fiber contraction which many times causes the viscus to become perforated? Spinal anesthesia should never be used in the presence of intestinal obstruction.

Time is again a potent factor when a complete laryngospasm occurs. When a laryngoscope or intratracheal tube is not available, or one feels he is not capable of using either, one can usually find a 2 cc. syringe and 1 to 2 cc. of water or saline. With a hypodermic needle through the neck into the trachea, the solution can be injected. The patient will then cough to expel it, thereby breaking his own laryngospasm.

Laryngeal edema in children seems to be of infrequent occurrence. In a recent conference of anesthesiologists from the Southern states, it was brought out that most

tonsilectomies are being performed under intratracheal anesthesia. It is easy to induce, rarely causes trauma, and offers a great deal more safety. With a free and clear airway, gastric dilatation rarely occurs, and with this avoided, another etiologic factor for a fatality is removed.

Again time is important if there has been a complete circulatory failure. Anesthesiologists should say to the surgeon, "Shall I open the chest, or will you?" As in the story we all know, "If you are going to argue about it, let's call the whole thing off."

One point that Dr. Carron did not have time to list is that blood introduced in the large arteries during massage of the heart will have a greater value than when introduced in the peripheral circulation. Many times there is not a sudden circulatory failure, for the process has been progressing for many minutes before failure occurs. The warning flags were not seen. Even at times we set the stage for this occurrence by the administration of too many drugs. I am a firm believer in the fact that with the increase in the number of drugs used there will be an increase in the number of complications.

Early in Dr. Carron's paper he spoke of neurogenic shock occurring with the stimulation under light anesthesia. This we are seeing with increased frequency, where anesthetists are guilty of substituting muscle relaxants, curariform drugs, for adequate anesthesia. It is an easy pitfall.

Again I should like to thank Dr. Carron for his contribution.

Cause and Treatment of Nonunion of Tibial Fractures

GUY A. CALDWELL, M.D.
NEW ORLEANS

Failure of union occurs more frequently in fractures of the tibia than of any other bone except the neck of the femur. The principal cause of tibial nonunion is imperfect immobilization of the fragments. Any new development, therefore, in the treatment of fractures which will provide better immobilization of the fragments should be employed in the treatment of tibial fractures if possible. Correct insertion of intramedullary nails in selected cases of tibial fractures seems to provide the required immobilization and thus should reduce the incidence of nonunion. Likewise, the better fixation of fragments provided by intramedullary nails is proving helpful in the treatment of cases of established nonunion of the tibia when combined with the application of suitable bone grafts.

The value of bone grafts to promote union is well established. Considerable discussion, however, has centered around the merits of various kinds of bone grafts, for example, autogenous or homogenous, cortical or cancellous, and massive or shavings or chips. Enough clinical experience in the use of these various kinds of grafts, as well as reliable experimental observations, has accumulated to indicate which bone grafts are preferable. This paper will review the subject of intramedullary fixation of tibial fractures and discuss the kinds of bone grafts that should be used in the treatment of nonunion of the tibia.

Intramedullary nails have been used most frequently and successfully to treat fractures of the shaft of the femur. Success has depended chiefly upon several principles. First, a long, rigid metal nail or tube can be safely inserted into the full length of the medullary canal of the femur and is well tolerated by the bone for many months. Secondly, such a rigid nail, passed through the full length of the medullary canal of the femur, provides almost perfect immobilization of fragments provided it fits snugly in the canal and extends far enough beyond the fracture line to secure a good purchase on each fragment. Finally, the internal fixation thus provided is so secure and strong that prolonged external fixation is unnecessary, muscles and joints may be exercised almost from the beginning, and weight bearing may be permitted before the callus has completely calcified. Union, therefore, is more certain, the periods of hospitalization and disability are shortened, and the possibility of muscle atrophy and stiffness of joints is diminished.

The same principles that have made the use of intramedullary nailing successful in femoral fractures are applicable to its use in tibial fractures. Actually, intramedullary nails are rarely employed for tibial fractures because the technical problems of introducing them are greater and somewhat different from those encountered in

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Read before the Florida Medical Association, Seventy-Ninth Annual Meeting, Hollywood, April 27, 1953.

femoral fractures. Retrograde insertion of an intramedullary pin in the proximal fragment of the tibia cannot be accomplished without opening the knee joint. This adds considerable risk to the procedure and probably should not be done except in highly selected cases. The method generally used requires the making of a slot in the inner tibial condyle through which the pin is directed down the canal and into the distal fragments. The hour-glass shape of the tibial medullary canal makes it impossible to have a nail or tube of uniform size fit snugly in the full length of the canal. Nested U-shaped pins or flexible nails therefore are required and when one of these has been successfully introduced, the tibial fragments are still not fixed as securely and certainly as are femoral fragments. Hence, there is need for external fixation with plaster for most of these fractures until early formation of callus takes place. In 1949 in a report on delayed callus formation in 8 of 56 cases of tibial fractures treated with intramedullary nails Lauritzen[1] wrote, "We can scarcely fail to lay the blame for delayed callus on the early weight bearing in these cases where the nail undoubtedly afforded insufficient fixation." Every effort, therefore, should be made to use pins which will provide maximum fixation and purchase on the fragments, and even when these seem to have been satisfactorily accomplished, it is probable that a long leg plaster should be worn for the first four to five weeks until the fragments are welded together with firm early callus. Then a walking plaster might be used, and, later, weight bearing without external support.

In relatively few cases of tibial nonunion, intramedullary nails have been employed for internal fixation in connection with bone grafting procedures. They have been so helpful in obtaining union in these cases, however, that it is probable they will be used more and more frequently as surgeons become more familiar with the exacting technic required for their insertion. The intramedullary pin provides firm fixation of the fragments in good position, leaving the entire circumference of the cortex available for the application of multiple bone grafts.

Nonunion with Loss of Substance

For nonunion of the tibia, especially when a defect of the bone exists, use of massive onlay grafts has been generally accepted as the method of choice. These grafts, consisting of the full thickness of the tibial cortex, are firmly attached to the tibial fragments with screws. In cases with defects more than 1 cm. long, dual grafts are often applied, one on either side of the bone. The rigid cortical bone, firmly attached with screws, has been considered essential to securing union because it seems to be the only way to provide complete immobilization and at the same time a stimulus to new bone formation. It has been generally recognized, however, that dense, cortical bone grafts are slowly replaced with new bone whereas cancellous grafts are replaced more rapidly.[2] When intramedullary pins are employed for fixation, cancellous grafts may be used to fill the bony defect or surround the line of nonunion; fixation with screws is not essential, thus avoiding additional foreign bodies and the trauma incident to their insertion; and external fixation may be discontinued sooner to permit exercise of muscles, motion of joints and weight bearing, all of which are essential to a good functional result. Despite the probable advantages of intramedullary nailing combined with the use of cancellous grafts for nonunion of the tibia, there will always be a certain number of cases, however, for which the onlay tibial grafts will be better adapted.

Separation of the tibial fragments of less than 1 cm. often results from early union of the fibular fracture, at full length, while gradual absorption of the ends of tibial fragments takes place, thus producing a gap which new bone cannot cross readily. In such cases it is essential that oblique osteotomy of the fibula be performed so that the tibial fragments can be forced together. This osteotomizing of the fibula is a necessary adjunct to other measures to promote union of the tibia, and one that should never be overlooked.

Delayed Union and Nonunion of Tibia With Good Apposition and Alignment

In many cases after three to six months' fixation in plaster, even if good position has been maintained, there will be little or no evidence of union, usually because of poor immobilization in plaster. In such cases it is desirable to take steps to promote more rapid union. Of the many methods that have been used, two are of proved value: (1) multiple drill holes crossing the fracture site, and (2) application of onlay grafts without screw fixation as advocated by Phemister.[3]

Multiple drill holes crossing the line of fracture obliquely from various directions permit ingrowth of blood vessels and stimulate repair. This procedure can be accomplished through a minimal

exposure and is followed by little or no reaction, swelling or pain. After the drill holes have been made, a snug, long leg plaster should be applied.

Phemister[3] proved that it is not essential to expose and freshen the ends of fragments and ream out the marrow cavities as was generally done before grafts were applied. When the fragments are well apposed and the alignment satisfactory, it therefore suffices to freshen and level one or two surfaces of the tibia across the fracture line and lay bone grafts alongside in contact with the raw surfaces. Phemister[3] found that screw fixation is not essential and that multiple short cortical or cancellous grafts serve to hasten callus formation and solid union. Osteotomy of the fibula is often required in addition, and application of a well fitted, long leg plaster is generally essential.

Selection of Grafting Materials

Many articles have appeared advocating homogenous bone grafts preserved by chemicals or by freezing, and others have emphasized the advantages of cortical grafts as opposed to those of cancellous bone. This disagreement has served to confuse the average reader. Doubtless each kind of graft has a field of usefulness; certainly no one type is universally applicable. Enough experience has now accumulated, however, to justify certain criteria in the selection of grafting materials: autogenous grafts are more certain to survive and promote osteogenesis than homogenous bone; cancellous grafts are more readily revascularized and replaced than cortical grafts and therefore usually result in more rapid union; fresh autogenous grafts are more likely to survive and less prone to produce infection than homogenous grafts preserved by freezing or in chemical solutions, although the latter may be used in selected cases; cortical grafts are most useful when strength and rigidity are of primary importance, and their osteogenic power can be enhanced by the addition of cancellous bone; and cancellous grafts are preferable whenever bone repair or bone replacement is delayed or absent.

Conclusions

1. Intramedullary nailing together with suitable bone grafts is an effective method of treatment of nonunion of the tibia.

2. Intramedullary nails supplemented by cancellous bone grafts are applicable to some cases of tibial nonunion with loss of substance which ordinarily are treated with massive onlay grafts.

3. Osteotomy of the fibula is a useful adjunct in the treatment of tibial nonunion with slight distraction of fragments.

4. Delayed union or nonunion of the tibia with good appositon of fragments usually will respond to small onlay grafts applied without screw fixation.

5. Autogenous grafts are preferable to homogenous grafts, although the latter may be used when the former are not available.

6. Cancellous grafts are replaced with callus more rapidly than cortical grafts, but the latter are essential when the problem requires strength and rigidity.

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Prytania and Aline Streets.

MEDICAL DISTRICT MEETINGS

Tallahassee, Oct. 19, 1953

Tampa, Oct. 21, 1953

St. Augustine, Oct. 20, 1953

West Palm Beach, Oct. 22, 1953

Congenital Malformations of the Thoracic Aorta

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The subject of malformations of the thoracic aorta encompasses mainly three categories of conditions: (1) patent ductus arteriosus; (2) aortic coarctation, and (3) the so-called vascular rings. The discussion of these conditions is important since each condition may lead to untimely death, and, at the same time, many patients with one or another of these malformations are candidates for relief or cure by surgical means.

Patent Ductus Arteriosus

In the fetus the ductus arteriosus is a vital channel carrying blood ejected from the right ventricle into the descending aorta from which vessel it flows to the placenta for exchange of oxygen and other substances. Normally, after birth the ductus arteriosus closes, at first, in all probability, by spasm. Later the lumen of the vessel becomes closed anatomically. Usually by a month after birth anatomic closure is complete. In some instances the anatomic closure is accomplished within two weeks from the time of birth.

If the lumen of the ductus arteriosus remains persistently patent, an abnormal shunt occurs. After birth the pulmonary arterial pressure normally is lower than the aortic pressure. Under these circumstances the flow of blood through the ductus arteriosus is in the reverse direction from that which occurs in the fetus. In the postnatal state the flow is from aorta into the pulmonary arterial system.

The shunted blood, which is already completely saturated with oxygen and represents about half of the average output of the left ventricle, then flows through the lungs to the left side of the heart and to the aorta, where the shunt is repeated. The right ventricle does not carry any of the blood involved in the shunt.

Under ordinary circumstances the burden of the shunt is carried by the left ventricle, which becomes dilated and hypertrophied. The left ventricle may ultimately fail, and this complication may in turn be followed by right ventricular failure. It must be emphasized, however, that failure of the left ventricle may be associated with death from pulmonary edema without right ventricular failure existing.

In addition to one or another manifestation of cardiac failure, the patient with a persistently patent ductus arteriosus is subject to the possibility of bacterial intravascular infection. When this occurs, it involves primarily either the ductus arteriosus itself or the major pulmonary arteries opposite the mouth of the ductus where the lining is traumatized by the jetlike stream of the shunted blood.

The two major complications of patent ductus arteriosus, namely, cardiac failure and bacterial infection, usually do not occur until adult life. It must, however, be emphasized that, although there is this delay in the majority of cases, left ventricular failure has occurred during infancy as a result of persistent patency of the ductus arteriosus. Furthermore, it must be emphasized that unless surgical closure of the ductus arteriosus is carried out in infants who have this complication, death during this early period of life may be caused from the resulting pulmonary edema.

It is axiomatic that the so-called machinery murmur of patent ductus arteriosus does not occur during the first few years after birth. Failure to realize this situation may result in overlooking the possibility of patent ductus arteriosus when in fact that condition represents the problem in a given case. In infancy the murmur of patent ductus is usually systolic and has a tendency to be loudest over the basal portion of the heart, although this localization of the murmur cannot always be demonstrated.

The history of the patient who is suffering from the effects of left ventricular failure incident

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to patent ductus arteriosus is not specific and may be duplicated by other conditions in which left ventricular failure plays a role. Some of these conditions are: ventricular septal defect, coarctation of the aorta, endocardial sclerosis, anomalous origin of the left coronary artery from the pulmonary trunk, subaortic stenosis, mitral insufficiency and paroxysmal tachycardia. Many patients have histories of poor feeding, failure to gain weight and attacks of "bronchitis" or "pneumonia." During these attacks cyanosis, which is not present between attacks, may be present. In all probability the attacks which are attributed primarily to pulmonary infection represent basically attacks of acute left ventricular failure with associated pulmonary edema. Careful examination and treatment are urgent for such patients because death may occur unexpectedly during an attack of pulmonary edema.[1-3]

In the ordinary case of patent ductus arteriosus the adult or adolescent patient has a pulmonary pressure within the range of normal. The circulatory disturbance in such cases has already been reviewed. A few patients having patent ductus arteriosus may have elevated pulmonary pressures which far exceed the normal range and approach or equal systemic pressures. In such cases a significant burden is placed on the right ventricle manifested by roentgenologic and electrocardiographic characteristics of right ventricular hypertrophy as well as by poor tolerance for exercise on the part of the patient. Since the volume of the shunt through the ductus arteriosus is in part dependent on the degree of difference in pressure between that in the aorta on one hand and that in the pulmonary arteries on the other, the magnitude of the shunt tends to fall when the pulmonary pressure rises. In extreme cases the direction of the shunt may be reversed with venous type blood flowing from the pulmonary arterial system into the aorta. In cases in which the pulmonary pressures are significantly elevated and the volume of the arteriovenous shunt is decreased, the left ventricle is proportionately relieved of the burden of the shunt. On the contrary the burden of the right ventricle is increased, and the chances of right ventricular failure become great.

The factors responsible for significant elevation of the pulmonary arterial pressure which occurs in the minority of patients having patent ductus arteriosus are probably several. They include organic occlusive lesions in the pulmonary vascular bed, functional response of the pulmonary vascular bed, excessive flow of pulmonary blood and left ventricular failure.

The outlook for an essentially normal cardiovascular system after surgical interruption of the ductus arteriosus is less certain for the patient having patent ductus arteriosus complicated by pulmonary hypertension than for the patient having an uncomplicated case of patent ductus arteriosus. Indeed, some wonder whether closure of the ductus arteriosus results in a fall in pulmonary pressure of sufficient degree to relieve the burden on the right ventricle. While this is still an unsettled problem as regards all cases, the experiences of several at the Mayo Clinic have been that in cases of pulmonary hypertension with patent ductus arteriosus ligation of the ductus arteriosus may, in some cases, be followed by significant reduction in pulmonary arterial pressure.

Aorticopulmonary Septal Defect

Aorticopulmonary septal defect is characterized by a window-like communication between the ascending aorta and the pulmonary trunk.[4] Though rare, this condition is to be considered in the differential diagnosis of patent ductus arteriosus since the circulatory abnormalities of the two conditions are essentially identical. Especially is the diagnosis of aorticopulmonary septal defect to be considered when a murmur similar to the machinery murmur of patent ductus arteriosus is heard best at some focus removed from the left subclavicular region.

Although in isolated cases the defect has been closed surgically, as yet a standard operative procedure has not been devised for the closure of this abnormal communication.

Aneurysm of an Aortic Sinus (Sinus of Valsalva)

Aneurysms of an aortic sinus usually involve the right aortic sinus and less commonly the posterior sinus.[5] These aneurysms may communicate with either the right atrium or ventricle from before birth or they may rupture secondarily into either of these cardiac chambers at some time after birth.

If a congenital communication exists between the right side of the heart and the aorta, a continuous murmur, usually loudest in the right side of the precordium, may be heard at the time of the patient's birth.

When an aneurysm of the aortic sinus ruptures some time after birth, the patient's history is usually one of well being until sudden onset of dysp-

nea and of a continuous precordial murmur. Although a communication between the aorta and a right-sided cardiac chamber is not amenable to surgical treatment at the time of this report, such a condition enters into the clinical differential diagnosis of patent ductus arteriosus because of the continuous nature of the precordial murmur.

Coarctation of the Aorta

In coarctation of the aorta, the malformation is one of the tunica media of the aorta. The basic narrowing is not caused by fibrous tissue; it is a true deformity of the aortic wall involving all parts of the media except that of the lower portion. There is a thickening of the media which causes profound narrowing of the lumen.

In classifications of coarctation of the aorta reference frequently is made to the so-called infantile and adult types. These terms are not useful. A classification which takes into account the questions as to whether the coarctation is proximal or distal to the ductus arteriosus and also whether the ductus arteriosus is patent or closed is preferable.[6,7] Accordingly, the following classification of aortic coarctation is offered:

A. Coarctation of aorta distal to ductus arteriosus
   1. With ductus arteriosus closed
      a. With branches of aortic arch normal
      b. With stenosis or atresia of origin of left subclavian artery
      c. With stenosis of origin of right subclavian artery
      d. With origin of right subclavian artery beyond coarctation as fourth branch of aorta
   2. With ductus arteriosus patent

B. Coarctation of aorta proximal to ductus arteriosus
   1. With ductus arteriosus closed
      a. Coarctation distal to origin of left subclavian artery
      b. Coarctation proximal to origin of left subclavian artery
   2. With ductus arteriosus patent

Coarctation is considered to be formed during fetal life, and the development of collateral vessels during this period depends on the position of the aortic narrowing with respect to the ductus arteriosus. Whether or not collateral vessels develop will be important to the patient after birth and after the ductus closes. If the coarctation is distal to the ductus, the normal fetal flow from ductus into the descending aorta is impossible, and the collateral vessels develop if the fetus is to survive and to get blood from the heart to the placenta. Coarctation beyond the ductus is the commonest variety. In this type of case collateral vessels have a chance to develop in fetal life, and the patient may be reasonably well off after birth when the ductus closes.

If the coarctation on the other hand lies proximal to the ductus arteriosus, the fetal circulation is disturbed little, if at all, since the blood from the right ventricle and ductus can readily enter the descending aorta. It is much like a normal fetal circulation. The stimulus for collateral development is deficient, and few or no collateral channels develop to by-pass the coarctation.

The clinical manifestations of coarctation vary greatly depending on whether the coarctation is distal or proximal to the ductus. In the usual type of coarctation the aortic narrowing is distal to the ductus, and the latter is closed. The branches of the aortic arch are normal. The patients in cases of this type have hypertension demonstrated by elevated brachial blood pressure, with absent or weak femoral pulsations. An aortic diastolic murmur may be present.

The extensive collaterals may be apparent as abnormal arterial pulsations in the interscapular regions. The same feature may be demonstrated roentgenographically by notching of the ribs. The latter sign, although frequently searched for if the patient is suspected of having coarctation, is not invariably present in adult patients and is usually absent in children. The electrocardiogram may help in some cases, but may not be an aid to diagnosis. Left ventricular strain may be apparent.

Coarctation distal to a closed ductus arteriosus but with associated abnormalities of the branches of the arch presents interesting clinical pictures. These are manifested by inequality in the force of the radial pulses and of the brachial blood pressures. When the left subclavian artery is either atretic or stenotic, the left radial pulse is absent or weak and the left brachial pulse usually cannot be measured with the ordinary blood pressure cuff, while hypertension and forcible pulses may be demonstrated in the right arm. If notching of the ribs is evident in the roentgenogram, this feature is limited to the right side. The femoral pulses are weak or absent. An identical picture is found in those cases in which the coarctation is proximal both to the ductus arteriosus and to the left subclavian artery and in which the ductus

arteriosus is closed.

When coarctation is present distal to the closed ductus and associated with stenosis of the right subclavian artery, there is likewise inequality in the pulses and blood pressures of the upper extremities, but the picture is the reverse of that of the case wherein the left subclavian artery is narrow. Origin of the right subclavian artery distal to the coarctation produces a clinical picture like that in the patient with coarctation and with stenosis of a normally arising right subclavian artery.

Coarctation of the aorta distal to a patent ductus arteriosus is associated with well developed collaterals. The individual patient presents the clinical manifestations commonly associated with aortic coarctation and patent ductus arteriosus. As a consequence of the greatly increased flow through the shunt, occlusive vascular lesions develop in the lungs and eventually evidence of patent ductus arteriosus complicated by pulmonary hypertension may be added to the clinical makeup of the patient.

In general, patients with coarctation distal to the ductus arteriosus, whether the ductus remains open or becomes closed, live longer than patients in whom the coarctation is proximal to the ductus arteriosus. Even the patients who have a better prognosis usually do not reach a full span of life since they are faced by a number of potential complications.

Cardiac failure resulting from the existing hypertension is one of these complications.[8] In some patients cardiac failure may be caused by insufficiency of a coexisting bicuspid aortic valve. This valvular malformation may be found in about three fourths of the patients with coarctation. In some of these, bacterial endocarditis of the aortic valve may be superimposed. Another complication that occurs in patients with coarctation of the aorta is a tear in the proximal aorta, a typical dissecting aneurysm of the aorta which usually leads to hematopericardium and death. Occasionally a patient having coarctation also has a saccular aneurysm beyond the coarctation. The occurrence of aneurysms in the circle of Willis is common in cases of coarctation, and in an occasional patient one of these may rupture, leading to subarachnoid hemorrhage. Dilatation of the anterior spinal artery may occur since that artery takes part in the collateral circulation. Rarely thrombosis of the anterior spinal artery with typical anterior spinal artery syndrome may occur.

As stated previously, when the coarctation lies proximal to the ductus arteriosus, collateral vessels which would by-pass the coarctation develop poorly or not at all. If the ductus remains open, the descending aorta is supplied by the right ventricle. Thus, only part of the right ventricular blood flows to the lungs; the remaining amount enters the descending aorta. The blood which enters the descending aorta re-enters the right ventricle after it has traversed the systemic circulation of the lower part of the body. The left ventricle is excluded from carrying the blood which supplies the part of the body below the level of the coarctation. Clinically the patients in cases of this type have good femoral pulsations, but the lower portion of the body may be cyanotic, since it is supplied with venous type blood. The clinical picture is entirely different from that in the usual type of coarctation. Functional disturbances result from the existing pulmonary hypertension and right ventricular strain.

When coarctation is proximal to the ductus arteriosus and the ductus closes, all of the blood which leaves the right ventricle traverses the lungs and is then expelled by the left ventricle. In the presence of inadequate collaterals, the coarctation may be considered as a significant barrier to emptying of the left ventricle, resulting in left ventricular strain and even left ventricular failure.[7,9-11] Such problems are observed in the newborn. As a rule the clinical manifestations of left ventricular failure in such cases do not differ from those of left ventricular failure resulting from simple patent ductus arteriosus or other conditions already listed. Usually there is roentgenologic and electrocardiographic evidence of left ventricular hypertrophy. Evidence of right ventricular hypertrophy may coexist. An important sign leading to the correct diagnosis is absence of femoral arterial pulsations. Kirklin and associates[12] have reported the case of a 10 week old infant in whom successful resection of the segment of aorta containing the coarctation and performance of an end to end anastomosis of the aorta were accomp'ished with disappearance of evidence of coarctation and of left ventricular failure.

Vascular Rings

The so-called vascular rings comprise a large number of vascular patterns of the aorta and its branches which collectively have the characteristics of producing undue pressure against the esophagus alone or in association with the trachea. Detailed descriptions of the characteristics of each of these

vascular configurations are in the literature.[13] Some patients having these abnormalities live a full life with no symptoms referable to the vascular malformation. If symptoms are present, they usually appear in infancy or childhood. Two categories of disorders are manifested, one resulting from esophageal obstruction, the other from tracheal obstruction. Esophageal obstruction causes regurgitation of food and even liquids in most severe cases. Tracheal obstruction is manifested by cough, at times brassy in character, wheezing and retraction of the head. At times hyperflexion of the spinal column is evident. In cases of severe tracheal obstruction inspiratory intercostal and supraclavicular retraction may be present. Pulmonary infection is common and may be the terminal complication. In some cases pulmonary infection constitutes the chief noticeable disorder, the patients being subject to recurrent attacks.

Since all of the vascular rings cause esophageal compression, whether producing symptoms of this disturbance or not, the simplest method of establishing the diagnosis of a vascular ring is to obtain an esophagram. If the condition exists, evidence of the deformity will be apparent in the upper portion of the thorax, usually at about the level of the third thoracic vertebral body. Bronchoscopic examination may reveal pulsating narrowing of the trachea, but this procedure is not as a rule necessary for the establishment of diagnosis of a vascular ring. Exact prediction of the anatomic nature of the malformation in a given case requires familiarity with the roentgenographic characteristics of the mediastinum. Treatment of patients having symptoms is surgical and is directed toward dividing those structures which would release the trachea and esophagus from the constricting effect of the vascular malformation, at the same time preserving vital vessels.[14]

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MEDICAL DISTRICT MEETINGS

Tallahassee, Oct. 19, 1953
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West Palm Beach, Oct. 22, 1953

Any member interested in presenting a scientific paper at one of these meetings is urged to make application without delay to Dr. John D. Milton, 701 Huntington Bldg., Miami, or to any Councilor, or an officer of your county medical society.

You may either speak in your own district or as a guest essayist in some other district, if you prefer.

Miliaria: Pathogenesis and Treatment

A Review of Recent Advances

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The seemingly simple disease called prickly heat has in recent years been the subject of much experimental work and thought. This interest has been the result of intensive studies by dermatologists in connection with military action in tropical theaters of war. It is timely that the physician in this semi-tropical climate be made familiar with the recent progress in this field.

Incidence

Contrary to popular belief, there is no greater susceptibility of the blonde, fair-skinned or red-headed person than of the brunette. Also, obesity does not predispose to greater incidence of the disease.[1]

Relation to Salt Intake

With such a high proportion of the population on a salt-poor diet for various cardiovascular conditions, it is interesting to note the results of Fay.[2] A low or moderate salt intake, up to 14 Gm. daily, helps to avoid miliaria, whereas a large intake of over 28 Gm. daily aggravates and predisposes to a greater incidence of the disease. It is suggested that this aggravation by a high salt intake is due to the subject's overcompensating for salt lost by sweating, with the result that there is an upset in the osmotic balance in the blood, followed by an increased rate of sweating.

Horne and Mole,[3] in studying this question of salt relation to miliaria, concurred in Fay's findings and found that increased intake of water relieved the aggravated miliaria.

There is evidence, summarized by Gregeisen,[4] which suggests that the skin can store sodium chloride, and with less than enough water to dilute it to isotonicity. This might explain why an increased salt intake shows itself in some circumstances as a cutaneous lesion.

The turnover of salt and water in a hot climate may be large and may vary considerably from day to day, depending on the environment and on physical exertion. Thus, short term alterations in salt and water balances can easily take place, and may be responsible for the great fluctuations in the incidence of miliaria.

Interestingly, O'Brien[5] emphasized in this connection that the anidrosis associated with miliaria itself leads to an alteration of salt and water balance.

For these reasons, the patient on a salt-poor diet, especially if he has a senile or ichthyotic skin, must be provided for in regard to prophylaxis in miliaria.

Local Cutaneous Edema in the Pathogenesis of Miliaria

Pollitzer,[6] in 1893, said: "This condition is evidently due to imbibition of the stratum corneum with water or with sweat. An epidermis which is bathed in perspiration, retained, as it is, by wet underclothing, cannot cornify; and, in imbibing water, the cells swell, and swell in all directions, laterally as well as vertically. Cells which are well impregnated with fat cannot imbibe water, and therefore will not swell up and occlude the sweat ducts when soaked with perspiration." He then cited the freedom of the well oiled Negro from miliaria and the toll of the disease among Englishmen in India who take "a couple of tubs" a day.

These opinions by Pollitzer[6] were lately confirmed in O'Brien's work. Typically, there is a lack of manifest sweating in areas of miliaria. This is not due to impairment or lack of sweat formation, but to failure to deliver the sweat to the surface of the skin. This failure is apparently caused by horny plugs in the sweat ducts and/or by inflammatory reactions and edema constricting the terminal part of the duct.

The Horny Plug in the Pathogenesis

The significance of the horny plug is explained in the theory of Pollitzer,[6] namely, that the keratin of the stratum corneum swells through imbibition and so blocks the mouths of the sweat ducts. He suggested that the keratin swells be-

cause it is not protected by sufficient lipoid. Lipoid depletion is probably not the sole or direct cause of miliaria as seen in the tropics. Nevertheless, it is probably a direct cause of poral closure in many ichthyotic skins.

Infection in the Pathogenesis

It is possible that temporary waterlogging of the pore, whether or not it produces closure, may pave the way for infection by staphylococci. Arnold[7] has shown that edema of the stratum corneum causes an increase in the endogenous bacterial flora of the skin. O'Brien,[5] in applying solid culture mediums to the skin, caused the formation of parakeratotic plugs in sweat pores as the result of experimental staphylococcic infection. He also found that gram-positive cocci are identifiable with considerable regularity in the pores in the earliest lesions in miliaria.

Where infection is absent, he found, edema and increased temperature are together capable of producing only a transient degree of poral closure, as manifested in the mild process called miliaria crystallina. Excessive soaping, too, causes parakeratotic plugging of the sweat pores, but the mechanism of this is unknown; whether it occurs by lipoid depletion, staphylococcus infection, or by direct injury is undetermined. Walker[8] found that the Staphylococcus aureus resists the antiseptic action of soap more than other bacteria, and therefore is still present after soaping, to play a part in causing miliaria.

O'Brien[5] carried out the following experiment to prove that the evil side effect of soap might be wrought, at least partly, through lipoid depletion. A soldier, with a history of a normal skin, soaped both sides of his abdomen for two minutes, five times daily. After each soaping, he applied a bland toilet powder to the test side, and then inuncted lanolin into the control side.

After one day, a miliarial rash was evident on the test side (with the powder), but the control side remained normal. Treatments were continued, and biopsy specimens taken from each side on the twelfth day. At that time, the test area had become relatively anhidrotic.

Keratotic plugs developed on the test side, and the control side remained normal. One may conclude that soaping with the ordinary alkaline soaps is contraindicated in the case of recurrent miliaria.

Summary of Treatment Aims and Methods

1. Inhibit excessive sweating		Cooler environment
2. Promote evaporation and drying	by	Reduce work load
3. Prevent and counteract maceration		Fewer clothes
4. Increase heat loss by other routes		Drink fluids
		Little salt intake
5. Diminish swelling of keratin		Producing superficial desquamation with
6. Prevent formation of horny plugs and remove horny plugs present	by	
7. Reduce the concentration of irritating agents in the sweat		℞ Menthol 1% Glycerin 1% Salicylic acid 4% Alcohol 95% qs Sig: Dab on affected areas several times daily
8. Allay the periductal inflammation		A full erythema dose of cold quartz light every three days
9. Prevent lipoid depletion, especially in senile skin		Avoiding soaps
10. Avoid irritation of skin	by	Use pHïsoHex® in cleansing once daily only
11. Avoid poral infection		Anoint with an antiseptic baby oil, after washing.

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American Building.

ABSTRACTS OF MEDICAL ARTICLES

Effective Treatment for Black Widow Spider Bite. By Edmonson S. Couric, M.D. South. M. J. 45:1193 (Dec.) 1952.

Three cases are added to the literature describing poisoning from the bite of Latrodectus mactans, the black widow spider. In these cases treatment with epinephrine administered subcutaneously brought prompt relief from the severe symptoms, which include nausea, vomiting, shock and extremely severe muscular cramps.

In the first case, treated in 1937, the patient experienced severe pain in the left eye in addition to the symptoms mentioned, which were extreme. After failing to respond to morphine and other therapy, he was in a moribund state with muscles drawn in knots and jaws locked when administration of 10 minims of epinephrine brought dramatic relief almost immediately. With each recurrence of muscular cramps this drug gave relief within three minutes. The sight of the left eye was not regained, however, and at present, after 15 years, he can barely distinguish daylight from dark because of an embolus. Whether the sight would have been saved had epinephrine been administered sooner remains problematic. In the second case 8 minims of epinephrine, and in the third case 7 drops, brought complete relief from the severe symptoms within six minutes, and no further treatment was necessary.

In reviewing the literature, the author was unable to find a report of a case in which epinephrine had been used in the treatment of black widow spider bite. The usual treatment described was calcium gluconate intravenously and morphine with recovery taking several days, and in some instances there was fatal termination.

The Dysenteries in the Armed Forces. By Albert V. Hardy, Richard P. Mason, and Gerald A. Martin. Am. J. Trop. Med. & Hyg. 1:171-175 (Jan.) 1952.

Certain aspects of an epidemic in Korea, involving prisoners of war, are presented as illustrative of the peculiar nature of the problem of dysentery in the Armed Forces. The magnitude of this epidemic is described as some 150 major epidemics compressed into one. Ordinarily, an outbreak of enteric infections with 161 hospitalized cases with a fatality rate of 9 per cent, and some 800 milder nonhospitalized cases, would be regarded as a major epidemic, but an average of this number of cases and deaths occurred daily for four months, with the outbreak continuing at a lower level for about a year.

The experience of the authors, members of the "Joint Dysentery Unit," a cooperative activity involving military and civilian organizations, including the Florida State Board of Health, led them to conclude: Epidemics of dysentery encountered in the Armed Forces may be of unprecedented size, the clinical and laboratory features may be atypical, and the opportunities for study may be unexcelled. There are simple though essential requirements for the handling of these major problems. For effective action, prompt reporting is as essential in military as in civilian practice. Consultative assistance in the clinical, laboratory and epidemiologic field may be required. A mobile laboratory may be essential for the highly important early studies of a major epidemic. Their experience in Korea directs attention to the value of the commissions of the Armed

Forces Epidemiological Board; a more frequent use of this available assistance in the field may be recommended. Certainly this study demonstrates how widely enteric infections encountered in the Armed Forces abroad may differ from those encountered at home.

Chronic Brucellosis, Recent Advances in Diagnosis and Treatment. By Lydia Allen DeVilbiss, M.D. J. Am. M. Women's A. 7:326-330 (Sept.) 1952.

The worldwide importance of brucellosis, as emphasized by the World Health Organization, is stressed in this paper. Methods of transmission, the author states, include the discovery that brucellosis is an air-borne infection; organisms will penetrate mucous membranes of the eyes, the respiratory and intestinal tracts, and will infect through the unbroken skin .

The characteristics of active chronic brucellosis and their similarity to those of tuberculosis are described. Also, methods of diagnosis, laboratory procedures and treatment are outlined. Twelve brucellosis research institutes in a worldwide distribution, where batteries of scientists are searching for improved methods of prevention, diagnosis and treatment, are mentioned as most hopeful features for the control of brucellosis, acute and chronic.

Pelvioprostatic Venography: Preliminary Report. By Raymond J. Fitzpatrick and Louis M. Orr. J. Urol. 68:647-651 (Sept.) 1952.

The authors present a preliminary report of the results of the injection of radiopaque materials into the deep dorsal vein in living subjects. In 5 cases they made pelvioprostatic venograms in an attempt to evaluate the usefulness of the procedure from the clinical standpoint. They conclude that it is difficult to be certain at the present time of the value of this procedure from the standpoint of adding further information to the diagnostic armamentarium. Until a sufficiently large number of pelvioprostatic venograms is obtained to established the normal pattern, the departure from the normal cannot be appreciated. They plan future studies to ascertain whether or not there is change of pattern of the vessels in cases of prostatic carcinoma with extension beyond the capsule and to test other uses of the procedure which immediately suggest themselves.

Surgical Considerations in Removal of Renal and Ureteral Calculi, with Special Emphasis on Removal of Staghorn Calculi. By S. Joseph Pearlman, M.D. Am. J. Surg. 84:302-305 (Sept.) 1952.

In view of the present widespread use of extensive nephrotomy in cases of staghorn calculosis, the author re-emphasizes some of the undesirable aspects of this operation and presents certain experiences with more conservative maneuvers which have yielded more satisfactory results. He discusses general principles to be observed in the removal of renal and ureteral calculi, and their importance to the prevention of recurrences. His preference is for the use of ureterolithotomy and pelviolithotomy, and even for ureterolitholapaxy, rather than extensive pole-to-pole nephrotomy, and he recommends more widespread use of direct roentgenologic control at the time of operation to reduce the incidence of "false" recurrences.

Syphilitic Optic Nerve Atrophy Treated with Penicillin: Observations Two to Six Years After Treatment. By Curtis D. Benton, Jr., M.D., and J. Frank Harris, M.D. A.M.A. Arch. Ophth. 48:449-454 (Oct.) 1952.

The results of this study indicate that penicillin is effective in arresting the early stages of atrophy of the optic nerve. Twenty-three patients with progressive syphilitic primary atrophy of this nerve were treated with one course of penicillin and observed for an average of four years after therapy.

There was satisfactory arrest of visual loss in all of the 7 patients whose initial visual acuity was 20/50 or better. In these cases the effective dose of penicillin was 4,800,000 to 6,000,000 units. All but 3 of the 16 patients whose vision was worse than 20/50 failed to retain their same vision, despite treatment with 6,000,000 to 18,000,000 units of penicillin. This critical level of pretreatment vision is apparently of prognostic significance, the authors conclude, and corresponds closely to the figure of 20/60 given by Moore as an important factor in predicting the outcome of malaria therapy.

It is noteworthy that when diminution of vision progressed, it usually did so within one and one-half years. It remains to be determined whether larger doses of penicillin alone or combined with fever will be beneficial to patients with moderate or severe visual damage.

Cutaneous Papillomatosis, Pseudo-Acanthosis Nigricans, and Benign Acanthosis Nigricans. By Morris Waisman, M.D. South. M. J. 46:162-169 (Feb.) 1953.

Review and comparison of *papillomatose confluente et réticulée* and pseudo-acanthosis nigricans lead the author to conclude that these two dermatoses are identical diseases, and he prefers the name pseudo-acanthosis nigricans. Occurring in obese and brunette persons and resembling acanthosis nigricans except for the reticulated configuration on the trunk, the disease is provoked by perspiration, maceration and friction, and it may disappear after reduction of weight.

Benign acanthosis nigricans, a genetically transmitted nevoid disturbance of the skin, is morphologically indistinguishable from the malignant form of acanthosis nigricans which is invariably accompanied by internal cancer. Clinically, pseudoacanthosis nigricans can be differentiated from acanthosis nigricans chiefly by the absence of a reticulated pattern of lesions in the latter. Whether a distinction can be made histopathologically is as yet inconclusive.

Since the sites on which lesions of pseudo-acanthosis nigricans localize are also those commonly affected by miliaria and intertrigo, it is suggested that in some way the activity of sweat glands may play a part. Diminution or absence of sweating over affected areas has been recorded, and decrease or absence of sweat glands is an almost constant histopathologic observation. It is conjectured that a variety of external influences, physical, chemical, and actinic, may lead to reversible papillomatous changes in the skin. The tendency to attribute the dermatosis of benign acanthosis nigricans and the associated obesity and dermatosis of pseudo-acanthosis nigricans to endocrine disease is unsupported by clinical and laboratory proof.

The Early Recognition and Treatment of Bronchogenic Carcinoma. By Jack Reiss, M.D., F.C.C.P., George L. Baum, M.D., and Maurice Kovnat, M.D. Dis. of Chest 22:529-538 (Nov.) 1952.

Since most patients with bronchogenic carcinoma reach the surgeon in an inoperable state, the authors stress the importance of recognizing the disease in the "silent phase." In this stage of bronchial cancer, the tumor may be evident but symptoms are not yet present. Pneumonectomy at this point should give far better results than are now obtained. They therefore stress the need for mass survey films.

In the series of 70 consecutive cases of bronchogenic carcinoma analyzed, only 20 were deemed operable. At operation, in but 10 of the 20 was the tumor resectable; of these 10, in only 5 had the patient survived for an average of 17 months from the earliest clinical manifestations of the disease. In 19 cases there was coexistent chronic pulmonary disease. During the period in which the 70 patients with bronchogenic carcinoma were admitted, only 23 with gastric carcinoma were similarly hospitalized. Seven illustrative cases are reviewed.

It is proposed that all adult members of the population have roentgenograms of the chest at six month intervals and that each survey film be inspected by two competent physicians, one of whom should be a qualified radiologist. The conclusion is that despite diagnostic advances, sole reliance on accepted principles of symptomatology and investigative technic causes the immediate goal of saving the lives of the majority of patients with this condition to fall far short of attainment.

Sexual Precocity: Case Report. By Richard G. Skinner, Jr., M.D. South. M. J. 46:194-195 (Feb.) 1953.

Sexual precocity, a relatively uncommon disorder, is classified by Wilkins as of four types: (1) neurogenic; (2) idiopathic activation of pituitary; (3) gonadal; and (4) adrenal. Dr. Skinner presents the case of a 7 year old white boy as an example of the type due to the idiopathic activation of the pituitary.

This case was regarded as one of the so-called constitutional type of sexual precocity because of the normal ketosteroid findings. If the precocity had been due to an adrenal tumor, the ketosteroid excretion should have been much higher, perhaps 15 to 30 mg. The presence of symmetrically enlarged testicles would tend to eliminate gonadal tumors, such as an interstitial cell tumor or teratoma. This also helped to rule out adrenal tumors, in which cases one usually finds infantile testicles. Lack of history of previous illnesses would tend to rule out encephalitis. Normal neurologic examination and roentgenograms of the skull helped in eliminating brain tumors.

An Introduction to the Problem of the Alcoholic. By Leonard L. Weil, M.D. South. M. J. 46:189-190 (Feb.) 1953.

The problem of alcoholism is approached from the standpoint of etiologic factors creating the situation, the treatment of acute alcoholic intoxication, and the treatment of withdrawal symptoms or the hangover. The reasons for drinking, as stated by the patients, are described as endogenous and exogenous and are discussed together with various theories. The present scientific approach to therapy is reviewed, including the conditioned-reflex-aversion method, which, regardless of how utilized, should be combined with psychotherapy, a psychologic survey, and sociologic adjustment. The therapeutic measures employed in the treatment of the hangover are described as limited only by the number of hangovers and all unsatisfactory.

Since the ever-increasing number of alcoholic addicts usually seek the aid of the family physician, the problem of alcohol addiction singularly falls in the realm of general practice. Emphasis is placed upon the responsibility resting upon the general practitioner. He has need to evaluate ever more carefully the problems of humanity for "it is his understanding of human frailty, his sympathy for his fellow man, his understanding of the problems that beset the species homo sapiens, that make his shoulders broad enough to sustain him who cannot sustain himself."

Trichomonas Vaginalis Donne: An Evaluation of Experimental and Clinical Data. By Carl Henry Davis, M.D., and C. G. Grand, M.D. Am. J. Obst. & Gynec. 64:544-551 (Sept.) 1952.

Regarding Trichomonas vaginalis as a possible contributing factor, through chronic irritation, in the development of cancer of the cervix uteri, the authors report a comparative study of slides in New York and Miami indicating that fewer women have a T. vaginalis infection in the latter area. They summarize experimental data and clinical observations and comment upon the ease of diagnosis. In discussing the problems of treatment, they observe that many of the numerous types of therapy tested have effected cures in 80 to 90 per cent of cases within a few weeks or months. While no drug has proved to be specific, practically all of the antiseptics in common use readily kill the T. vaginalis. It is their observation that vaginal preparations containing aureomycin hydrochloride, recently recommended by some clinicians, have not provided a higher percentage of apparent cures than may be obtained by other available preparations and that the severe yeast infection which may follow the use of this antibiotic may be more difficult to cure than that caused by T. vaginalis.

Today there is better understanding of the nature of infection with this organism and greater appreciation of the need for daily treatments over a variable period of time. Also, newer pharmaceuticals have facilitated the necessary home treatments. Nevertheless, it is concluded that probably just as high a percentage of cures may be obtained by the use of ichthyoglycerine tampons and medicated douches at home, containing older drugs such as mercury bichloride, formalin, liquor cresolis, tincture of green soap, potassium permanganate, or iodine in appropriate strengths. Also, silver nitrate commonly has been used in office practice. All of these, with the possible exception of ichthyol, have been demonstrated to be among the most effective trichomonacides.

The Henry Approach to Femoral Hernia. By Harry C. Hull, M.D., and Joseph B. Ganey, M.D. Ann. Surg. 137:57-60 (Jan.) 1953.

The anterior, low midline extraperitoneal approach as described by Arnold K. Henry in 1936 is advocated for the repair of femoral hernias. The technic is described. A series of 16 cases is reported with case reports of 2 fatal cases.

The authors favor this approach for several reasons. It allows an excellent and easy approach to unilateral or bilateral femoral hernias through one incision. Their elective repair can be made in conjunction with other abdominal and pelvic operative procedures. If an anomalous obturator artery is present, it is readily discernible and can be avoided, or, if necessary, safely ligated. Cooper's ligament is accurately defined and the obliteration of the femoral canal can be made with this ligament or pectineal fascia as snugly as desired without danger to the iliac vein, which is at all times under direct vision.

Also, the hernial sac and contents can be released by incising the lacunar and/or inguinal ligaments under direct vision. Abundant access to a wide area of parietal peritoneum is obtained so that complete inspection and necessary definitive surgery can be performed with ease. The use of this approach in the obese patient is, however, not advised.

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From Our President

Giving Versus Receiving

The rewards for attention to a medical practice in these days are all too often measured in terms of money and financial security. While it is true that we are engaged in the practice of a profession or business, for which we are recompensed to a greater or lesser degree, and that the possession of some money is necessary if we are to continue to practice our profession and carry on our businesses, there are other compensations which are not measurable by conventional standards. But these other compensations, arising in the course of a medical practice, depend on the exercise and demonstration of those qualities which endeared the old time family physician to his patients and friends. The time just to talk, the hearty handclasp, the cheering word, the friendly smile, the outpouring of friendship, characterized the family physician of yesteryear.

You cannot receive to the fullest extent unless at the same time you are giving to the extent of your capacity. If this is a selfish concept, it is still fundamental. People are and will be interested in you to the same degree in which you are interested in them, and manifest that interest to them. It has been said that the esteem of the physician is no longer what it was, and that his stature has diminished in the eyes of the public. It is strange, is it not, that the advances in surgical and medical skill, new technics, new drugs, innumerable new discoveries and advancing knowledge of disease have not counteracted this tendency to loss of professional prestige in the eyes of those we serve? If this is so, it has been of our own choosing. There is more to the practice of medicine than surgical skill, knowledge of new drugs and how to use them, advanced methods of diagnosis and improved methods of administering treatment, and the introduction of modern business methods and practices in the doctor's office and in the hospital and clinic.

We possess, just as our forefathers in medicine possessed, those characteristics which endeared them to their patients. We can share of our personality, as they did of theirs, manifesting a wholesome interest in the affairs of the individual, the locality in which we live, our state and nation, contributing generously and enthusiastically to the well-being of those with whom we come in contact, sharing with them our enthusiasm for medicine, our confidence in America and its institutions. We have much to share. Let us be generous while we can.

Frederick K. Herpel

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Outlook for Polio Protection

There is probably no subject today on which the physician is more frequently interrogated than that of poliomyelitis. At this writing the quest for adequate protective measures against this crippling disease has turned the nation's eyes hopefully on Montgomery County, Alabama, where a mass polio inoculation project with gamma globulin is under way.

In a final report evaluating gamma globulin as a prophylactic agent for poliomyelitis, Hammon and his associates[1] concluded that in extensive field tests on numerous children gamma globulin in an average dose of 0.14 cc. per pound of body weight conferred highly significant protection against paralytic poliomyelitis for a short period of time. They emphasized that gamma globulin is no panacea for the prevention of the paralytic form of polio, pointed out its drawbacks and disadvantages as well as its advantages, and stressed that it should be given intramuscularly, never intravenously, and with a separate heat-sterilized syringe and needle for each child. This report merits the physician's careful perusal.

For the present and immediate future, it appears that gamma globulin from blood is the only means of protection, aside from that "touch of polio" which is either not noticed in children or recognized only as a slight feverish upset, but is effective enough to cause polio antibodies to be formed. The future, however, gives promise of an effective, safe vaccine from such sources as monkey kidneys and fertile hen's eggs.

Salk and his associates[2] recently reported studies in human subjects inoculated with different experimental poliomyelitis vaccines. For the preparation of these vaccines virus of each of the three immunologic types was produced in cultures of monkey testicular tissue or monkey kidney tissue. Prior to inoculation of human subjects, the virus was rendered noninfectious for the monkey by treatment with formaldehyde. In one series of experiments antibody for all three immunologic types appeared to be induced by the inoculation of small quantities of such vaccines incorporated in a water-in-oil emulsion. In other experiments, intradermal inoculation of aqueous vaccines containing the type 2 virus induced formation of antibodies which persisted without signs of decline for four and a half months, the longest interval studied. It was concluded that it should be possible with a noninfectious preparation to approximate the immunologic effect induced by the disease process itself.

These encouraging results point to a new source of protective vaccine, the supply problem for which can be solved. One pharmaceutical

manufacturer plans to make the vaccine available immediately after it has passed all tests for safety and effectiveness. He thinks the cost will not be prohibitive, even with monkeys at $45 to $50 apiece already arranged for from India, because of the good yield of virus for vaccine from the tissues of the monkey kidney.

Fertile hen's eggs are under investigation as a new and readily available source of supply in the field of poliomyelitis virus cultivation. For those authorities who believe that a killed virus never gives as lasting protection as a live one, this source has particular appeal. Cox and his associates[3] have grown the polio virus on fertile hen's eggs, and tests show that the virus is truly modified, causing formation of protective antibodies but not causing polio. They were able, however, at last reports to get only one strain of one type of polio virus to grow on this medium, while all three types will grow on monkey kidney and are included in the vaccine developed by Salk and his associates.[2] The egg vaccine not only solves the supply problem, but it has the additional advantage of being given by mouth. No injection is necessary, and in Dr. Cox's opinion the route of administration is advantageous because it follows the natural way the polio virus enters the body, through the mouth and the digestive tract.

These tests offer encouragement in the fight against polio which may mean another triumph for medicine is at hand. Meanwhile, the research data presented on gamma globulin should help the practicing physician to use the material in the most effective manner and aid him in keeping such records as will be of greatest value in the search for more knowledge about the effective use of gamma globulin.

1. Hammon, W. McD.; Coriell, L. L.; Wehrle, P. F., and Stokes, J., Jr.: Evaluation of Red Cross Gamma Globulin as a Prophylactic Agent for Poliomyelitis: 4. Final Report of Results Based on Clinical Diagnosis, J. A. M. A. 151: 1272-1285 (April 11) 1953.
2. Salk, J. E., and others: Studies in Human Subjects on Active Immunization Against Poliomyelitis: 1. A Preliminary Report of Experiments in Progress, J. A. M. A. 151:1081-1098 (March 28) 1953.
3. Blood '53 Anti-Polio Hope, Science News Letter 63:230 (April 11) 1953.

1953 Medical District Meetings

"A", Tallahassee	October 19
"B", St. Augustine	October 20
"C", Tampa	October 21
"D", West Palm Beach	October 22

Body Appreciation

There are music appreciation classes and literature appreciation classes. Why not body appreciation classes? The idea seems constructive. Every doctor-patient interview might well become such a class, to the benefit of patient and physician alike.

Physicians as well as patients are prone to take the body for granted. That veritable treasure house grows commonplace as patient after patient drones on, reciting trivial or imaginary or easily avoided ills. But really serious ailments come as sharp reminders that humanity is fearfully and wonderfully wrought. From a physical standpoint, each one of us is a marvelous creature. If that fact were more fully appreciated and the body treated with more respect and consideration, the harassed physician would have more time to devote to combating serious diseases and would find the practice of medicine more stimulating. In the interest of body appreciation, he might impress upon his patients how richly endowed they are. He might say:

Take the very hairs of your head, for example. You doubt that they are numbered? Ask the Federal Bureau of Investigation. There you can be identified by one single hair, an unmistakable mark of your individuality, made for you alone.

Where do you find perfection in cameras? They are standard operating equipment right in your own head, set for a lifetime of service if given reasonable care. Two of them, just to make sure, adjust to light and dark, take pictures in black and white and in full colors, even in full motion, and develop them instantaneously. These miniature cameras which are your eyes are such wonderful bits of creative genius that they enable you, without changing the lens, to look at yourself or the stars in the heavens innumerable light years away.

The ears, two again for good measure, are minute sensory organs, really private, personal radios, which solve difficult problems of mechanics and organize a complex variety of air waves into meaningful patterns. Their performance is truly awe-inspiring. Yet how often their protection is neglected, especially by the swimmer.

Is the skin just a covering to hold you in, or is it a remarkable sensory organ whose highly refined sensitivity is a most precious possession? Surely many of you delight to abuse it in the Florida sun.

Think how poverty-stricken you would be if your brain were any less versatile. For example, take the gift of memory, of reasoning, of judgment, of ability to solve problems. Perhaps the greatest abuse of the brain is failure to develop its potential capabilities.

What a collection of factories the body has to furnish nourishment, repair and growth. The bellows that keeps the oxygen flowing makes of your lungs a complete blood-purifying factory. The factories of the alimentary tract convert raw material into nutritive elements. The sugar factory in your mouth, the chemical plant which is your stomach, the intestinal workshop, all carry on their complicated tasks of selection, assimilation and waste disposal, no matter how much or how often you insult them. The kidneys and other glandular bodies also carry on their intricate functioning as they secrete and stop secreting according to some master plan. And where is the electronic system which can compare in performance to your nervious system?

Skeleton? Who wants a skeleton? But what a sight you would be without one. The one you have is exceedingly durable, withstands shock, and even repairs itself from within. It gives you protection where you need it without hampering action. Nevertheless, sheer carelessness on your part takes an excessive toll in fractures.

All these rich and highly individual endowments, including not only the power of repair but also that of reproduction, are of no avail without that faithful little pump which keeps your blood circulating from before birth even until death. It is estimated that your heart will beat 800 million times if yours is a normal life span. This one pound muscle does an amazing amount of pumping just to keep you going a single day. Yet you not only take your heart for granted, but you keep disease of this organ in the forefront of causes of death by thoughtlessly and persistently abusing it.

So, "Have a heart," as the saying goes. Take up body appreciation as a new interest and reap untold benefits.

That goes for you, too, Doctor. You are quite as marvelous a creature as is your patient.

The Editor Invites Your Contributions on

Data of Notable Interest

World Medical Association Important to All Physicians

What the World Medical Association is and what it stands for are matters of moment to every physician. Too often it is confused with the World Health Organization, which is a branch of the United Nations, representing the governments of the world in the field of health and supported entirely by government funds. The World Medical Association, on the other hand, is an organization of national medical associations, composed of one from each of the participating nations which is most representative of the medical profession of that country. The American Medical Association is the American member. Thus this international body represents the practicing profession of the world and is supported entirely by dues and voluntary contributions.

Why should the average doctor have any interest in this organization? "First of all," says Dr. Louis H. Bauer, secretary-general of the World Medical Association and immediate past president of the American Medical Association, "an increasing number of problems relating to health and medicine are being discussed and decided on an international level. Most of the bodies discussing these problems have one viewpoint only, and that is the viewpoint of government. Without the World Medical Association, there is no one to present the opinions of practicing physicians. Their views should be heard and considered, but they cannot give them as individuals. Their voice can be heard only through an organization. The World Medical Association, through its 43 member associations, can speak for 700,000 physicians. I believe all will agree that this is a voice that should not be ignored. Furthermore, what happens in other countries is sooner or later likely to affect us here. Therefore, we should work with our colleagues in other countries and attempt to solve the problems facing the profession.

"Six years of experience with doctors of other nations has shown me conclusively that the same problems face the profession in all countries. They differ only in degree. That, perhaps, is not surprising. What is surprising is that doctors all over the world think alike about these problems, differing only as to details."[1]

Out of his wide experience with physicians abroad, Dr. Bauer has often emphasized how fortunate this country is that government has not

made the inroads on the practice of medicine that it has in many other countries. He points out that one way to keep medical practice free is to cooperate with other countries and fight the encroachments of socialism by the international route. In this respect, the contribution of the World Medical Association alone in exposing the machinations of the International Labor Organization[2] should command the support of the physicians of this country. Sponsorship of the First World Conference on Medical Education, held in London last April, is another great contribution of the World Medical Association to the welfare of medicine. Additional examples of its importance are its studies of the medical aspects of social security, standards of hospitals and development of an international code of medical ethics, its stand on an international pharmacopeia, and its continuing battle to protect the rights and privileges of doctors in both peace and war.

Supporting committees have been established in some countries in which individual membership is possible. In the United States, the World Medical Association, United States Committee, Inc., is a nonprofit organization which medical societies, business organizations and individuals may join. Members receive the association's publications, which keep them abreast of medical news of the world; they may attend the general assemblies of the association as official observers; and they may receive letters of introduction facilitating visits to medical centers in other countries.

The ability of the World Medical Association to speak for the practicing profession is its greatest function. It too often happens that the profession is ignored when matters of moment to both the profession and the public are decided. Here is an effective voice in international medical affairs, long greatly needed. And here is a great opportunity for all doctors to help make the World Medical Association a powerful influence in molding world opinion about medicine.

1. The President's Page, J. A. M. A. 151:1494 (April 25) 1953.
2. The Bricker Resolution, editorial, J. Florida M. A. 39:766-767 (April) 1953; ILO — Danger Ahead! editorial, ibid. 39:278 (Oct.) 1952.

First Global Conference on Medical Education

A history-making medical conference on a global basis will take place this month. The First World Conference on Medical Education will be held at the British Medical Association House in London, August 22-29. Among the 600 physicians attending from all over the world will be representatives from practically all of the 72 approved medical schools in the United States.

Sponsoring the conference is the World Medical Association, which has a membership of national medical associations from 43 countries. Cooperating agencies are the World Health Organization, the Council of International Organization of Medical Sciences, and the International Association of Universities.

Practicing physicians from most of the countries of the world will have an opportunity at this meeting to state how medical education has met or failed their needs. The entire subject will be reassessed in the light of present day advances in medical science, and a free exchange of ideas will be encouraged throughout the conference.

Many of the physicians attending from this country will go on to The Hague for the Seventh General Assembly of the World Medical Association, scheduled for August 31 through September 7.

DOCTOR, ARE YOU MOVING?

Keep The Journal advised as to your current address.

University Honors Doctors

At its Centennial commencement on June 8, 1953, the University of Florida signally honored four Florida physicians. Upon Drs. William C. Thomas, Sr., of Gainesville, Walter C. Payne, Sr., of Pensacola, Turner Z. Cason of Jacksonville and Warren W. Quillian of Coral Gables it conferred the honorary degree of Doctor of Science. All of these distinguished physicians of the state have long been prominently identified with the Florida Medical Association, and Drs. Thomas and Payne are past presidents. Their activities in promoting numerous aspects of public health and medical education and their constructive approach to medical problems have made Florida a better state in which to live.

Dr. Thomas, beloved citizen of the University community, was honored for his contributions to medical science at home and abroad and for his unselfish devotion to the highest traditions of the medical profession which have elevated him to a place of leadership among the medical practitioners of Florida.

Recognized as a consecrated practitioner of a noble profession for more than 40 years, Dr. Payne was honored for his labors both as a physician and developer of hospitals and health services in the state, a contribution matched by a record of public service in other fields involving human welfare.

Native son of the University city, Dr. Cason was honored as an indefatigable advocate of medical education and adequate health services for the people of Florida and for his unceasing efforts, now crowned with success, to develop a great medical center at the University, where the youth of Florida may learn the healing arts.

Dr. Quillian was accorded recognition as a specialist in pediatrics, medical administrator and friend of children and was honored for contributions to the alleviation of human suffering so numerous and outstanding as to merit the praise and gratitude of his adopted state.

Doctors labor much more diligently in the public interest than the public realizes. The man

Left to right, Drs. Walter C. Thomas, Sr., Warren W. Quillian, Walter C. Payne, Sr., and Turner Z. Cason.

in the street would doubtless be astonished to learn the extent to which they freely give of themselves in professional services rendered without compensation and in time spent without remuneration in the interest of the public weal. If these services could be valued accurately, we doubt not that some critics of the medical profession would be truly astonied — probably to the point of incredulity.

It is, therefore, particularly heartening when a great state institution elects to honor physicians for especially meritorious service. Such gracious acts redound to the good of the profession as a whole and enhance its prestige in the public eye.

A. M. A. Annual Meeting in New York

In characteristic fashion, New York City extended its famed hospitality to the members of the American Medical Association and their guests who in record numbers attended the 102nd annual meeting of that organization there the first week in June. The magnitude of the convention is reflected in the official total registration of 48,980, including guests and exhibitors, the largest in the association's history and doubtless a world record for a medical meeting. This figure, broken down to 17,958 physicians, 24,186 residents, interns, technicians, students, nurses and physicians' guests, and 6,836 exhibitors and their guests, is in sharp contrast to an attendance of 573 when the association first met in New York City exactly a century ago. Some 5,000 more physicians were in attendance than when the annual convention was last held there in 1940.

More than 400 papers were presented before the 21 scientific sections on clinical and experimental studies, reports of surveys, reports of new drugs and technics. An innovation was presentation for the first time of medical colored television projected on two large screens, the telecasts of clinics previously having been received on several small screens. There were panel discussions of the diseases involved before the operations began, and the diseases presented were mitral stenosis, cancer of the cervix, peptic ulcer, tumor of the breast, and sinusitis. In addition, more than 6,000 persons viewed a variety of medical motion pictures presented twice daily, often with the authors themselves present to discuss their own films.

More than a thousand physicians and scientists gave voluntarily of their time and energy to demonstrate 260 scientific exhibits, in which emphasis was placed on aspects of medicine of particular interest to the general practitioner. A special attraction was the exhibit based on a survey of 400 general practitioners showing the instruments and drugs considered most essential for a doctor's bag to enable him to handle almost any emergency. The scientific exhibits occupied the entire fourth floor of the Grand Central Palace, and on the first three floors 370 technical exhibits were arranged with some 2,000 persons assigned to answer questions about drugs, surgical and diagnostic instruments, appliances, and many other products. Here was a vast "medical merchandise mart" displaying innumerable new items from which physicians could select those most helpful in their daily practice.

The House of Delegates, meeting at the Waldorf-Astoria Hotel, took important policy actions on veterans' medical care, medical ethics, osteopathy, intern training and a wide variety of other subjects which ranged from public relations to medical education.

Among the distinguished personalities particularly in the limelight were the incoming President, Dr. Edward J. McCormick of Toledo, Ohio, and Dr. Walter B. Martin, President-Elect. A Virginian by birth, Dr. Martin has a brilliant record of service to medicine during the 35 years he has devoted to the practice of internal medicine in Norfolk in his native state. Highlighting the recent progress in heart surgery was the award of the Distinguished Service Medal of the American Medical Association to Georgia-born Dr. Alfred Blalock of Baltimore. A pioneer in cardiac surgery, this internationally famous Southerner received the association's highest scientific honor for his outstanding work in vascular surgery, especially for his part in the development of the "blue baby operation," which has saved the lives of hundreds of infants with congenital pulmonary stenosis.

Featured entertainment was provided by the Doctors' Symphony Orchestra of New York, which received particular acclaim. Another highlight was the presentation of a citation to Jean Hersholt, the familiar "Dr. Christian," for his portrayal of the family doctor.

The immensity and complexity of the meeting, featuring a notable scientific program, important policy actions, exhibits, television shows, motion pictures, radio broadcasts, fraternity meetings and alumni reunions, made a profound impression on the nearly 49,000 physicians and

guests in attendance. Physicians at work and at play presented a unique display even for New York.

This year's Clinical Session will be held in St. Louis, and the next annual meeting will go to San Francisco. Florida will welcome the 1954 Clinical Session for Miami has been selected as the host city.

Registration

Total registration of Association members at the 1953 annual meeting in New York was 129. Members in attendance were:

BRADENTION: Lowrie W. Blake. CLEARWATER: John T. Karaphillis. COCOA: Thomas C. Kenaston. CORAL GABLES: Jack Q. Cleveland, C. Howard McDevitt, Jr., Frederick P. Poppe, Louis C. Skinner, Jr., Hillard W. Willis. DANIA: Fred E. Brammer. DAYTONA BEACH: John J. Cheleden. DELRAY BEACH: James R. Nieder. FORT LAUDERDALE: Norris M. Beasley, Russell B. Carson, Alfred E. Cronkite, Burns A. Dobbins, Jr., Richard A. Mills. FORT MYERS: Joseph D. Brown, Harry Fagan, Jr. GAINESVILLE: Alva T. Cobb, Jr., William C. Thomas, Jr. HIALEAH: Eli Galitz. HOLLYWOOD: Dale T. Anstine, William J. Gallo, Maxwell M. Hartman, Royle B. Klinkenberg, Bernard Milloff, Randall W. Snow. JACKSONVILLE: Jackson L. Allgood, Jr., William H. Brooks, Joseph A. J. Farrington, Leonard Garten, Gordon H. Ira, Samuel S. Lombardo, Bernard J. McCloskey, Robert B. McIver, Jerome H. Newman. KISSIMMEE: John O. Rao. LAKELAND: John E. Daughtrey, David S. Kenet. LAKE WORTH: Carl M. Pults. MIAMI: Lawrence Adler, Ernest R. Barnett, Gail E. Chandler, Reuben B. Chrisman, Jr., Carl H. Davis, M. Jay Flipse, Samuel Gertman, Herman Glassman, James J. Griffitts, Winston F. Harrison, Ella M. Hediger, Carlos P. Lamar, John J. McAndrew, Wayne B. Martin, Isidore Marx, Leon H. Mims, Jr., E. Sterling Nichol, Colquitt Pearson, Homer L. Pearson, Jr., Irwin Perlmutter, William C. Phillips, Edwin P. Preston, Hunter B. Rogers, Wiley M. Sams, George F. Schmitt, Jr., Robert T. Spicer, Donald G. Stannus, Richard F. Stover, Herbert W. Virgin, Jr. MIAMI BEACH: Bernhard Baer, Max Dobrin, I. Leo Fishbein, Abraham R. Hollender, Valentine E. Jenkins, Saul H. Kaplan, Harold S. Kaufman, Leo M. Levin, Alexander Libow, David S. Light, Meyer B. Marks, Saul Miller, Julius R. Pearson, Francis A. Reed, Maurice J. Rose, Jack A. Rudolph, Jandon Schwarz, Benjamin L. Steinberg, Nicholas A. Tierney. MILTON: John W. Reece. NAPLES: James A. Craig, John J. Meli. OCALA: William H. Anderson, Jr., Carl S. Lytle. ORLANDO: Benjamin L. Brock, Solomon D. Klotz, John G. Marsh, Pleasant L. Moon, Louis M. Orr, II. PALM BEACH: Fred E. Manulis, Joseph R. West. PENSACOLA: Herbert L. Bryans. QUINCY: George H. Massey. ST. AUGUSTINE: Vernon A. Lockwood. ST. PETERSBURG: Arnold S. Anderson, Arthur Appleyard, Jr., James K. McCorkle, Norval M. Marr, Richard H. Sinden. SARASOTA: John M. Butcher, Joseph Halton, Melvin M. Simmons. SEBRING: Leldon W. Martin, Zaven M. Seron. TAMPA: Frank S. Adamo, Harold O. Brown, Joshua C. Dickinson, Gaetano C. Ferrante, Nathan L. Marcus, Alfonso F. Massaro, Anthony P. Perzia, Burdette Smith, Harold Sutker, Wesley W. Wilson. VERO BEACH: John P. Gifford, James C. Robertson. WEST PALM BEACH: Harry E. Bierley, Theodore Norley, Ralph M. Overstreet, Jr. WINTER HAVEN: Henry F. Keiber.

Graduate Medical Education

The Seminar on Gastroenterology presented by the Department of Medicine of the Graduate School of the University of Florida, in cooperation with the Florida Medical Association and the Florida State Board of Health, was held at the George Washington Hotel in Jacksonville, June 18-20, inclusive.

Dr. H. Marvin Pollard, Professor of Internal Medicine at the University of Michigan, delivered the lectures on Thursday and Friday, and with the assistance of Dr. James L. Borland held a clinic and question period Saturday morning. The patients presented at the clinic provided clinical material illustrative of the lectures delivered the two previous days. This practical demonstration aided in stimulating many pertinent questions, both regarding the patients and the points brought out by Dr. Pollard. He was practical and lucid in the manner of presenting his subjects and at the same time brought postgraduate gastroenterology up to date for his listeners.

The Twenty-First Graduate Short Course for doctors of medicine was presented to the physicians of Florida at the George Washington Hotel in Jacksonville the week of June 22-27. Each subject was ably handled by the individual specialist.

This year's course was notable for the fact that each lecture carried with it information which the physicians in attendance were glad to receive. This appreciation was manifested by their interest throughout the whole course. Each course was presented by a well known teacher in his particular field of medicine. The excellency of the method of teaching was evidenced at each lecture.

The total registration for both the Gastroenterology Seminar and the Short Course was 212, a lamentably poor attendance when the facts are considered. The Committee on Medical Postgraduate Course met Wednesday, June 24, and will meet again in the fall to consider plans for graduate medical education in 1953-1954. It is highly probable that radical changes will be made next year in the methods of presenting what has been known in the past as the Graduate Short Course. This Graduate Short Course was designed for the general practitioner, and this idea has dominated the thinking in all the planning during the past 21 years. Apparently the lack of interest on the part of the general practitioner does not justify a continuation of this type of planning even though all graduate medical education presented under the auspices of the University of Florida in cooperation with the Florida Medical Association and the Florida State Board of Health is accredited and accepted by the American Academy of General Practice. Suggestions offered prior to the October meeting of the Committee will be welcomed and given serious consideration.

STATE BOARD OF HEALTH

Maternal and Child Death Rates — 1952

Members of the medical profession and public health personnel concerned with maternal health are to be commended for the large drop in maternal mortality in this state during 1952.

Analysis of the preliminary data on maternal deaths for 1952 shows that the maternal death rate decreased from the previous low record of 12.1 maternal deaths per 10,000 live births in 1951 to a new low of 7.7 in 1952. This represents a decrease of 36 per cent since the preceding year. The rates for both white persons and Negroes showed about the same percentage of change.

Table 1 compares the 1952 preliminary data with 1951 data. It is noted that the rate for Negroes of 16.4 is almost four times as high as the rate of 4.3 for white persons.

Table 1.—Comparison of 1952 Preliminary Data with Data for 1951

Race	Maternal Deaths		Death Rate*	
	1952	1951	1952	1951
Total	57	85	7.7	12.1
White	23	35	4.3	7.0
Negro	34	50	16.4	25.1

*Deaths per ten thousand live births.

A comparison of maternal mortality for Florida and the United States indicates that the Florida maternal death rate for 1952 is a little higher than the national average. When rates for two separate geographical locations are compared, however, a compensation should be made

Table 2.—Recorded Maternal Deaths by Cause of Death and Race, Florida, 1951 and 1952

Cause of Death	Total		White		Negro	
	1952	1951	1952	1951	1952	1951
Total all causes	57	85	23	35	34	50
Sepsis of pregnancy	0	0	0	0	0	0
Sepsis of childbirth and puerperium	7	15	3	8	4	7
Toxemia of pregnancy	13	24	2	5	11	19
Toxemia of the puerperium	7	2	4	1	3	1
Hemorrhage of pregnancy	0	1	0	0	0	1
Hemorrhage of childbirth	6	13	2	7	4	6
Abortion without sepsis or toxemia	3	1	0	0	3	1
Abortion with sepsis	3	7	2	2	1	5
Ectopic pregnancy	2	9	0	4	2	5
Other complications of pregnancy, childbirth and the puerperium	16	13	10	8	6	5

for the difference in proportion of nonwhite births. When the Florida maternal death rate is adjusted to compare with the national figure, it turns out to be 6.1, or exactly the same as that for the United States. It is hoped that for future years the Florida rate will be lower than the national average.

Table 2 indicates the number of maternal deaths according to cause for the years 1951 and 1952. The largest decreases were noted in sepsis of childbirth and puerperium, toxemia of pregnancy, hemorrhage of childbirth, and ectopic pregnancy.

While great strides were made in reducing maternal deaths, the infant death rate rose. From table 3 it can be seen that the rise was primarily due to an increase in the deaths of Negro infants.

Table 3.—Comparison of 1952 Preliminary Data with Data for 1951

	Infant Deaths		Death Rate	
Race	1952	1951	1952	1951
White	1399	1362	26.2	27.0
Negro	1149	967	55.6	48.4
Total	2548	2329	34.4	33.1

The following comments concerning Negro infant deaths give some indication of where future efforts should be placed. Largest increases were in the diseases listed:

Cause	1952	1951
Postnatal asphyxia and atelectasis	148	89
Pneumonia	171	134
Nutritional maladjustment	29	18
Ill-defined diseases of early infancy	58	47
Gastritis, duodenitis, enteritis and colitis	79	69
Congenital malformations	63	55

Deaths from infections and parasitic diseases dropped from 52 to 37.

Deaths from postnatal asphyxia and atelectasis were predominantly in the group under 4 days of age.

Deaths from pneumonia were predominantly between the ages of 14 days and 9 months.

Infant mortality rates increased in 44 counties.

All age groups under 9 months of age showed an increase in death rates with the greatest increase in those under 1 month.

BIRTHS, MARRIAGES AND DEATHS

Births

Dr. and Mrs. Sanford Levine of Miami Beach announce the birth of a son, Herbert Neal, on May 16, 1953.

Dr. and Mrs. Edward E. Cava of Miami announce the birth of a daughter, Anita Ada, on May 26, 1953.

Dr. and Mrs. Frank L. Fort of Jacksonville announce the birth of a daughter, Mildred Leslie, on June 11, 1953.

Dr. and Mrs. David B. Manley of Orlando announce the birth of a son, David Bott III, on June 19, 1953.

Marriages

Dr. Harry Kaufman of Miami Beach and Miss Francine Abigail Sontag of Kew Gardens, N. Y., were married on June 13, 1953.

Dr. Bernard S. Kleinman of Miami Beach and Miss Helen Ratkay of Chicago were married on June 14, 1953.

Deaths — Members

Smith, H. Mason, Tampa ..... June 16, 1953

Deaths — Other Doctors

Futch, T. Allen, Jr., Thomasville, Ga. ..... March 20, 1953
Gonzalez, Aurelio A., Tampa ..... June 13, 1953

NEW MEMBERS

The following doctors have joined the State Association through their respective county medical societies.

Lacy, George E., Miami
Lansman, Wilfred, Miami Beach
McDermid, Howard C., Okeechobee

Medical Officers Returned

Dr. Thomas W. Meldrum, who entered military service on July 1, 1951, was released from active duty on April 16, 1953 with the rank of lieutenant (U.S.N.). His address is 8630 Lem Turner Road, Jacksonville.

STATE NEWS ITEMS

Dr. Sherman B. Forbes of Tampa attended the annual meeting of the American Ophthalmological Society in Hot Springs, Va., the last week in May.

Dr. Raymond J. Fitzpatrick announces the opening of his office at 926 S. W. 2nd Avenue, Gainesville, for the practice of urology.

Dr. Walter H. Winchester of Dunedin entered medical service with the U. S. Navy on May 31, 1953 with the rank of lieutenant.

Dr. Douglas R. Murphy of Venice entered medical service with the U. S. Navy on May 1, 1953 with the rank of lieutenant.

Dr. Joseph D. Foley of Jacksonville entered military service on May 4, 1953 as a captain in the Air Force.

Dr. Terry Bird of Apalachicola recently visited the Association's headquarters office.

Dr. Meredith Mallory, Sr., of Orlando, member of the Board of Governors, was a recent visitor to the Association's headquarters office.

Dr. J. Basil Hall of Tavares recently addressed the Kiwanis Club of Groveland. He spoke on the subject of individual health and prolonging the life span.

Dr. Richard C. Cumming of Ocala was recently re-elected president of the Marion Players of that city.

Dr. Herbert L. Bryans of Pensacola recently addressed the Hospital Trustee Institute at Panama City. His subject was "Trustees and the Medical Staff."

Dr. Jack H. Bowen of Jacksonville took a week's course at the Skin and Cancer Hospital in New York City in May.

Dr. Carl J. Larsen of Avon Park recently addressed the Rotary Club of that city. His subject was "Killer Disease."

Dr. Peritz Scheinberg of Miami recently addressed the Dade County Chiropody Society.

Dr. Charles R. Wiley has opened offices for medical practice in Englewood.

Dr. James N. Patterson of Tampa was installed as president of the Florida Association of Blood Banks at the recent meeting of the Association in Tampa. Dr. John T. Stage of Jacksonville was elected president-elect of the Association. Other officers installed included Dr. James M. McClamroch, Gainesville, vice president, and Dr. Sherman B. Forbes, Tampa, secretary.

Dr. Louis M. Orr, II, of Orlando was elected a member of the Council on Medical Service succeeding Dr. James Q. Graves of Monroe, La., at the recent meeting of the American Medical Association in New York City. The term runs until 1958. Dr. Orr also served as a member of the A. M. A. House of Delegates reference committee on Insurance and Medical Service.

Dr. Edward J. Clark of Callahan was a recent guest speaker at a meeting of the Forest-Dale Woman's Club of Jacksonville. He showed the film, "Self-Examination for Cancer," and conducted a question and answer period.

Dr. Thomas M. Palmer of Jacksonville attended a reunion of Rhodes Scholars in England in June.

Dr. Richard G. Skinner, Jr., of Jacksonville recently attended a Seminar on Rehabilitation of the Handicapped Child held under the auspices of the New York University Medical School, Bellevue Hospital.

The late Dr. Henry B. Cordes of Frostproof was recently honored by the Frostproof Garden Club, who chose Decoration Day for the planting of the Dr. Cordes memorial tree, a mahogany tree.

Dr. Grace C. Hardy of Jacksonville spoke on "Interdependence of Nations" at the Coronation Luncheon given by the Daughters of the British Empire recently. Dr. Hardy attended the Children's Conference in Miami in April. This Conference was sponsored by the Dade County Pediatric Society, the Nemours Foundation and the Florida Crippled Children's Society.

Dr. Bernard A. Kaminski of Miami Springs entered medical service with the U. S. Air Force on Nov. 8, 1952 with the rank of captain.

Dr. Morris A. Price of Jacksonville spoke at the Ramona Boulevard School summer roundup in May.

Dr. James F. Speers of Titusville was a recent guest speaker at a meeting of the Titusville Kiwanis Club.

WANTED — FOR SALE

Advertising rates for this column are $5.00 per insertion for ads of 25 words or less. Add 20c for each additional word.

WANTED: Affiliation with group, industry, sanatorium or hospital. Well trained and wide experience. Category IV. Prefer Fort Lauderdale, Florida area. Write 69-94, P. O. Box 1018, Jacksonville, Fla.

LOST: One old gold earring with small diamond during Annual Convention Hollywood Beach Hotel. Very valuable to owner. Reward. Write William H. Grace, M.D., 71 First Street, Fort Myers, Fla.

PSYCHIATRIST: Association with individual, group, clinic or location for private practice; diversified experience, ECT, child guidance, administrative, private practice, teaching. Board eligible, 18 years experience. Write 69-95, P.O. Box 1018, Jacksonville, Fla.

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COMPONENT SOCIETY NOTES

Broward

The Broward County Medical Society has announced the beginning of a transcribed radio program on station WBRD, Fort Lauderdale. Dr. Thomas L. Roberts, Jr., of Fort Lauderdale, chairman of the Society's Committee on Public Relations, is in charge.

Dade

The regular monthly meeting of the Dade County Medical Association was held on July 7. Following adjournment of the business meeting, there was a general discussion of medical economics problems.

Franklin-Gulf

The Franklin-Gulf County Medical Society met on June 3 at Beacon Hill.

Lake

Dr. Wilbur Davis of Orlando was guest speaker at the meeting of the Lake County Medical Society on June 3. His subject was "Some Problems in Oral Surgery."

At the July meeting of the Society, Dr. Newton C. McCollough of Orlando spoke on "Bursitis."

Marion

The Marion County Medical Society has paid 100 per cent of its state dues for 1953.

Orange

Drs. Francisco A. Hernandez, Ralph Edwards and Milton S. Saslaw of Miami were guest speakers at the meeting of the Orange County Medical Society on May 20. Dr. Hernandez spoke on "Catheterization in Congenital Heart Disease;" Dr. Edwards spoke on a "Children's Cardiac Clinic;" and Dr. Saslaw spoke on "The Rheumatic Problem in Florida."

Palm Beach

The Palm Beach County Medical Society in conjunction with the State Board of Health is sponsoring free tests for diabetes.

Taylor

The Taylor County Medical Society has paid 100 per cent of its state dues for 1953.

OBITUARIES

George A. Paulk

Dr. George A. Paulk died at his home in Coral Gables on Feb. 9, 1953, after a lengthy illness following surgery performed several years previously for carcinoma of the prostate. He was 76 years of age. Burial took place in his native town of Alapaha, Ga.

Dr. Paulk was born in Alapaha on Feb. 25, 1876. Following graduation from high school he went directly to the University of Louisville School of Medicine. On receiving his medical degree, he spent some time with the doctors of his community until he reached the age of 21, the minimum age required for the practice of medicine in the state of Georgia. Here, as is the custom with the country doctor, he took a most active part in community and business affairs. He was the organizer and president of the Alapaha Bank, in which capacity he served until coming to Miami in 1926. Subsequently, he moved to the larger neighboring town of Tifton where he continued in the general practice of medicine, which included surgery of the kitchen table variety.

In Tifton, Dr. Paulk continued to serve his community and state. He served in the Georgia Legislature first as Representative in the House in 1913-1914 and again as Senator from 1915-1916. In 1924-1925 he was Chief Industrial Commissioner and at various times served as chairman of numerous committees, namely of State Sanatoria, of Agricultural Commission, of Banks and Banking, of Education, of Hygiene, of Penitentiary Committee, of Rules Committee, of Western and Atlanta Railroad Committee, of University of Georgia Committee and of the Temperance Committee.

Before coming to Miami in 1926, Dr. Paulk became interested in Eye, Ear, Nose and Throat and took postgraduate work at Grady Hospital, New Orleans and a final six months' course at Polyclinic in New York. In Miami he associated himself in the practice of his chosen specialty with his brother-in-law, the late Dr. Austin Kemp. Following Dr. Kemp's death, he associated himself with the E. J. Hall Clinic of Miami. Here he continued to practice until several months before his death, during which time he was the oldest physician, in point of active practice, in Dade County. While during the last ten years Dr. Paulk's practice was confined to a full schedule of office work, he was at one time on the staff of Victoria Hospital and Jackson Memorial Hospital.

Dr. Paulk was a member of the Dade County Medical Association, the Florida Medical Association and the American Medical Association. He was a member of the Phi Chi medical fraternity and also a Mason and a Shriner.

Besides his widow, the former Doris Kemp, he leaves a son, George A. Paulk, Jr., of New York City, and two daughters, Mrs. W. I. Burt of Cleveland and Mrs. John Behney of Miami.

Luke Harvey Bartee

Dr. Luke Harvey Bartee of Port St. Joe died at the Municipal Hospital in that city on Feb. 16, 1953 after a long illness. He was 81 years of age. Interment took place in Magnolia Cemetery at Apalachicola.

Dr. Bartee the son of James Allen and Julia Ann Thornton Bartee, was born at Roxanna, Ala., on April 3, 1872. Early in his life his parents moved to Georgia, settling on a plantation in Meriwether County near Woodbury. Upon finishing his high school education there, he entered the University of Georgia School of Medicine at Augusta. In 1899 he was awarded the degree of Doctor of Medicine by that institution. He was licensed to practice medicine in Georgia that same year, and was licensed in Florida in 1905.

In 1920, Dr. Bartee located in Port St. Joe, where he continued in the general practice of medicine for 33 years. A devout Christian gentleman, he was a member of the First Baptist Church and taught the Men's Bible Class there for a long time. He was a member of the Masonic Lodge, both in Georgia and Florida.

Dr. Bartee was for some years a member of the Franklin-Gulf County Medical Society and of the Florida Medical Association. He also held membership in the American Medical Association.

(Continued on page 128)

Clinical Results* with Banthine® Bromide

(Brand of Methantheline Bromide)

22 Published Reports Covering Treatment of 1443 Peptic Ulcer Patients with Banthine

Comprising the reports published in the literature to date which give specific facts and figures of the results of treatment

AUTHORS	No. of Patients	Chronic, Resistant to Other Therapy	TYPES OF ULCERS				RELIEF OF SYMPTOMS (Chiefly Pain)				Surgery or Complications[1]	Side Effects Requiring Discontinuance of Drug[2]	EVIDENCE OF HEALING			
			Duodenal	Jejunal	Stomal	Gastric	Good	Fair	Poor	No Report			Complete	Moderate	None	No Report
Crimson, Lyons, Reeves	100	100	93	7			80	11	4		5		47		19	29
Friedman	15	15	14			1	5		4	6[3]			2			13
Bechgaard, Nielsen, Bang, Gruelund, Tobiassen	26	26	21			5	16	4	6				8	6	12	
McHardy, Browne, Edwards[4], Marek, Ward	162		162				136	12	11		3	1	14	9	7	129
Segal, Friedman, Watson	34	34	34[5]				14	13			7	2	5		8	14
Brown, Collins	117	99	117				97	7	8		5	8	55	9	8	40
Asher	77		65		7	5	52	9	16			16		9	21	47
Rodriguez de la Vega, Reyes Diaz	5	4	5				4		1					3	2	
Winkelstein	116	116	102	8		6	102		14				53		18	45
Hall, Hornisher, Weeks	18	18	18				11		1	6[3]			18			
Maier, Meili	38	38	24			14[6]	27	7	4[7]				10	2	5	21
Meyer, Jarman	25	18	25				21		4							25
Poth, Fromm	37	37	37				33	3	1				33	3	1	
Plummer, Burke, Williams	41	41	41				36		5				38		3	
McDonough, O'Neil	104	100	104				63	10	31			11	4		11	89
Broders	60	60	58		1	1	35	19	6				10	1	49[8]	
Legerton, Texter, Ruffin	11		11				11									11
Holoubek, Holoubek, Langford	76	69	76				35	27	10		4	10	26		10	36
Ogborn	42		39	2		1	42[9]									42
Shaiken	48	48	48				33	10	3		2		33	10	3	
Johnston	145	145	145				143		2			2	143		2	
Rossett, Knox, Stephenson	146		141			5	146					4[10]	53			93
TOTALS	*1443*	*968*	*1380*	*17*	*8*	*38*	*1142*	*132*	*121*	*12*	*26*	*54*	*552*	*52*	*179*	*634*
PERCENTAGES		*67.8*	*95.6*	*1.2*	*0.6*	*2.6*	*81.3*	*9.4*	*9.3*			*3.7*	*70.5*	*6.6*	*22.9*	

1. Not included in tabulations.
2. Included in "Relief of Symptoms" as "Poor" and in "Evidence of Healing" as "None."
3. Four had no symptoms when Banthine therapy was begun.
4. Of which seven were penetrative lesions and five partially obstructive.
5. No symptoms were present in four.
6. Two with symptoms only; no demonstrable ulcer.
7. Three were psychopathic patients and one had a ventricular ulcer of the lesser curvature.
8. Roentgen findings after treatment period of two weeks; forty-seven had duodenal deformity.
9. All returned to work within a week.
10. In these four, after relief of symptoms, Banthine was discontinued because of urinary retention.

During the past three years, more than 250 references to Banthine therapy in peptic ulcer and other parasympathotonic conditions have appeared in medical literature. Of these reports, 22 have presented specific facts and figures on the results of treatment in a total of 1,443 peptic ulcer patients, 67.8 per cent of whom were reported as chronic or resistant to other therapy. These results are tabulated above and show:

"Good" relief of symptoms was obtained in 81.3 per cent of the 1,405 patients on whom reports were available.

"Complete" evidence of healing was obtained in 70.5 per cent of the 783 patients on whom reports were available.

In all but 9.3 per cent, relief of pain was "good" or "fair." In all but 22.9 per cent, evidence of healing was "complete" or "moderate."

During treatment, 26 patients required surgery or developed complications other than ulcer which required discontinuance of the drug before results could be evaluated.

Of the remaining 1,417 patients, only 3.7 per cent experienced side effects sufficiently annoying to require discontinuance of the drug.

*Volume containing complete references, with abstracts of 39 additional reports, will be furnished on request by

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(Continued from page 126)

Survivors include the widow, Mrs. Esther Childers Bartee of Port St. Joe; one son, Edward Harvey Bartee, and one daughter, Mrs. Mae Bartee Dutton, both of Mobile; five sisters, Mrs. W. S. Sims and Mrs. J. L. Edwards of Manchester, Ga., Mrs. B. B. Lovett and Mrs. Rachel Sheppard of Woodbury, Ga., and Mrs. J. S. McCuffin of Jacksonville; and one brother, J. M. Bartee of St. Petersburg. Three grandchildren also survive.

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Walter Eugene Wilkins

Dr. Walter Eugene Wilkins of Jacksonville died suddenly at his home on March 2, 1953. He was 49 years of age. Funeral services and interment took place in Mars Hill, N. C.

The son of Walter and Sallie Bailey Wilkins, Dr. Wilkins was born on July 9, 1903, in Columbia, S. C. He received his academic training at Furman University and the University of North Carolina. In 1933 he was awarded the Ph.D. degree in biology and chemistry by Vanderbilt University, and in 1938 the School of Medicine of that institution conferred upon him the M.D. degree. From 1929 to 1936 he was an instructor at Vanderbilt, and in 1938-1939 served an internship in pediatrics at Vanderbilt Hospital. He then continued his training at Willard Parker Hospital in New York City during 1939.

Throughout the decade of the forties Dr. Wilkins engaged in public health activities. He served as director of the division of school health of the North Carolina State Board of Health from 1939 to 1942; medical nutrition officer of the War Food Administration in Washington from 1942 to 1945; senior surgeon (res.), United States Public Health Service, in charge of the nutrition section in 1945; and director of nutrition investigations and services in the Florida State Board of Health from July 1946 until he resigned in 1950. At that time he entered the private practice of medicine in Jacksonville.

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Dr. Wilkins was a member of the Duval County Medical Society, the Florida Medical Association and the American Medical Association. Since 1940 he had been a member of the governing council of the American School Health Association, serving as its president in 1946-1947. He was also a member of Phi Beta Pi and Alpha Omega Alpha.

His contributions to medical and scientific journals were numerous. He was the inventor of the cup needle for taking blood samples for laboratory tests.

Surviving are the widow, Mrs. Katherine Gibbs Wilkins of Jacksonville; two daughters, Mrs. Erik Tranoy of Oslo, Norway, and Miss Ann Kierulff Wilkins of Jacksonville; his mother, Mrs. Sallie B. Wilkins of Long Beach, Calif.; one brother, Dr. Franklin B. Wilkins of Whittier, Calif.; one sister, Mrs. Louise Leiby of Long Beach, Calif.; and an uncle, Col. F. N. K. Bailey of Sebring.

(Continued on page 130)

Cook County Graduate School of Medicine

POSTGRADUATE COURSES — 1953

SURGERY—Intensive Course in Surgical Technic, Two Weeks, starting September 14, September 28, October 12. Surgical Technic, Surgical Anatomy & Clinical Surgery, Four Weeks, starting October 26. Surgical Anatomy & Clinical Surgery, Two Weeks, starting August 17, November 9. Gallbladder Surgery, Ten Hours, starting October 26. General Surgery, One Week, starting October 5. Surgery of Colon & Rectum, One Week, starting September 21. Basic Principles in General Surgery, Two Weeks, starting September 21. Thoracic Surgery, One Week, starting October 12. Esophageal Surgery, One Week, starting October 19. Breast & Thyroid Surgery, One Week, starting October 26. Fractures & Traumatic Surgery, Two Weeks, starting October 26.

GYNECOLOGY—Intensive Course, Two Weeks, starting September 21. Vaginal Approach to Pelvic Surgery, One Week, starting August 31.

OBSTETRICS—Intensive Course, Two Weeks, starting October 5.

DERMATOLOGY—Intensive Course, Two Weeks, starting October 19.

MEDICINE—Electrocardiography & Heart Disease, Two Weeks, starting October 12. Intensive General Course, Two Weeks, starting September 28. Gastroenterology, Two Weeks, starting October 26. Allergy, One Month and Six Months, by appointment.

CYSTOSCOPY — Ten-Day Practical Course starting every two weeks.

UROLOGY — Intensive Course, Two Weeks, starting September 28.

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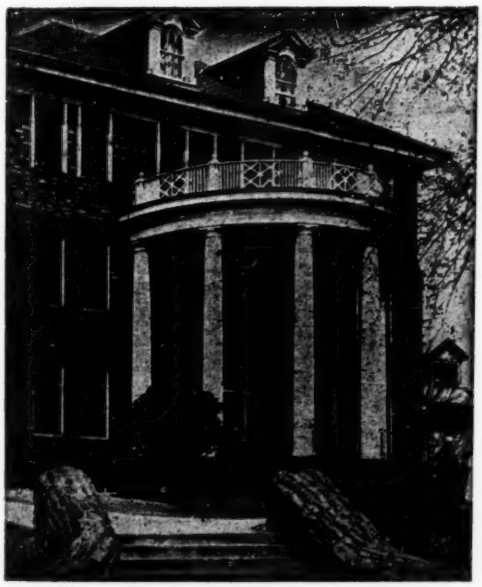

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Medical Director

ROBT. L. CRAIG, M.D.,
Diplomate in Neurology and Psychiatry
Associate Medical Director

(Continued from page 129)

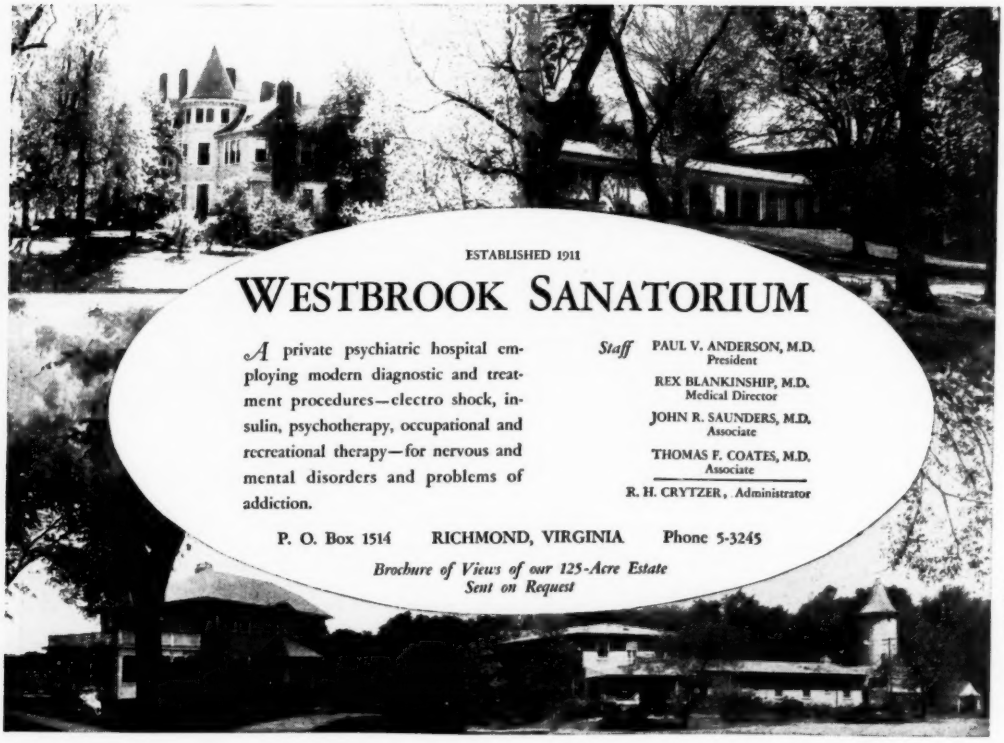
Thomas Joseph Moore

Dr. Thomas Joseph Moore of St. Petersburg died at Duke Hospital in Durham, N. C., on Feb. 21, 1953, after an illness of several months. He was 32 years of age. Funeral services and interment were in Elmira, N. Y.

A native of New York, Dr. Moore was born in Elmira and attended school there. He received the degree of Doctor of Medicine in 1945 from Duke University School of Medicine. After serving an internship at New York Post-Graduate Hospital, he entered military service in 1946 and was subsequently discharged at Bolling Field Station Hospital.

Dr. Moore had engaged in general practice in the St. Petersburg area for two years. His offices were at Madeira Beach, and he resided at Redington Beach. He was a staff member at both Mound Park and St. Anthony's hospitals in St. Petersburg.

A member of the Pinellas County Medical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Society, Dr. Moore had held membership in the Florida Medical Association since 1951. He was also a member of the American Medical Association.

He is survived by his widow, Mrs. Nancy Spangler Moore, and one daughter, Catherine Dale Moore, of Redington Beach; and his mother, Mrs. D. B. Moore of Bronxville, N. Y.

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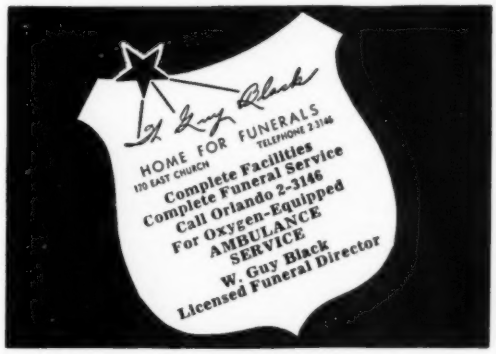

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Advertisement

From where I sit *by* Joe Marsh

It's Fine to Be Fooled —Sometimes

Handy Peters entertained for the ladies of the Auxiliary the other night —and had all the ladies believing for a while that he's the best marksman in the county.

Handy put on a great act. He set up a whole bunch of balloons on a muslin backdrop and then took out his pea-shooter. He shot blindfolded, standing on his head, every which way—and broke a balloon every time.

No wonder Handy made a big impression on the ladies. What they didn't know—till the show's end—was that Buck Mulligan was hiding behind the backdrop improving on Handy's aim with a hatpin.

From where I sit, we all get things "put over" on us now and again. When it's good-natured—fine! But, some folks would fool us into believing it's wrong to enjoy a glass of beer. Others would tell their neighbor how to practice his profession. For real American tolerance and neighborliness these people are simply "off target."

Joe Marsh

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Current Affairs is Your Affair

In pursuit of my favorite topic, education, I want to talk about Study Group, especially in Medical Auxiliaries. Study Group anywhere is valuable, but nowhere does self-improvement benefit so many as in medical circles. The simple reason is that the doctor and his family have been and will continue to be a target of interest as well as suspicion.

This singular position was brought about by an avalanche of adverse propaganda, conceived by politically active men who opposed the existing medical system and wished to replace it with a system foreign to our way of life. The ensuing battle has left its mark. The very victory we achieved through education, in itself, makes us more vulnerable, for in proclaiming that we have preserved an American way of life, we have given the people a right to expect us to understand and defend it.

We resisted a national law which by its passage in Congress would have deprived the American doctor of freedom in medical enterprise in accordance with the Constitution of his Association. We could not have done that without knowing why it was important to the medical profession as well as to the country. Nor could we have known without concerted study of the issues involved. We had to know in order to care

and we had to care in order to educate those who did not know or care, and by so doing we improved ourselves and preserved a precious freedom.

To do less when our nation's freedom is in danger of being legislated away is to lock one's self in a center room of a house which is catching fire.

For instance, in the April issue of The Journal, an editorial, "The Bricker Resolution," quotes another editorial entitled, "ILO — Danger Ahead!" It "pointed out that this nation is now under greater threat of socialism by treaty than by domestic legislation and urged widest recognition of the possibility of political and economic regimentation from external sources. It cited a treaty adopted last June by the ILO embodying socialized medicine, which requires only a two-thirds vote of the Senate for ratification, making it a law of the land regardless of the will of the people." Is this possible? What is the Bricker Resolution? How does it affect you? What is the ILO? How does it function? Does treaty agreement have precedence over national law? What are the external sources which threaten?

These questions and many more could be explained and understood with an active Study Group in an Auxiliary. It would make the doctor's wife a leading and constructive force in the community in which she so frequently takes an active part.

Before women became organized and earned the right to vote, it was not as dangerous for us to be ill-informed, but in an age when the voting potential of women can admittedly wreck a nation by voting its liberties out of existence, a greater obligation rests upon us to stress thinking in our groups.

When we stop to remember the number of bills we have been asked to endorse in various organizations to which we belong and remember the manner and method employed to achieve unanimous endorsement, it might be wise to consider the importance of thinking out a subject before we speak on it and to understand a resolution before we vote upon it.

Mrs. C. Robert DeArmas

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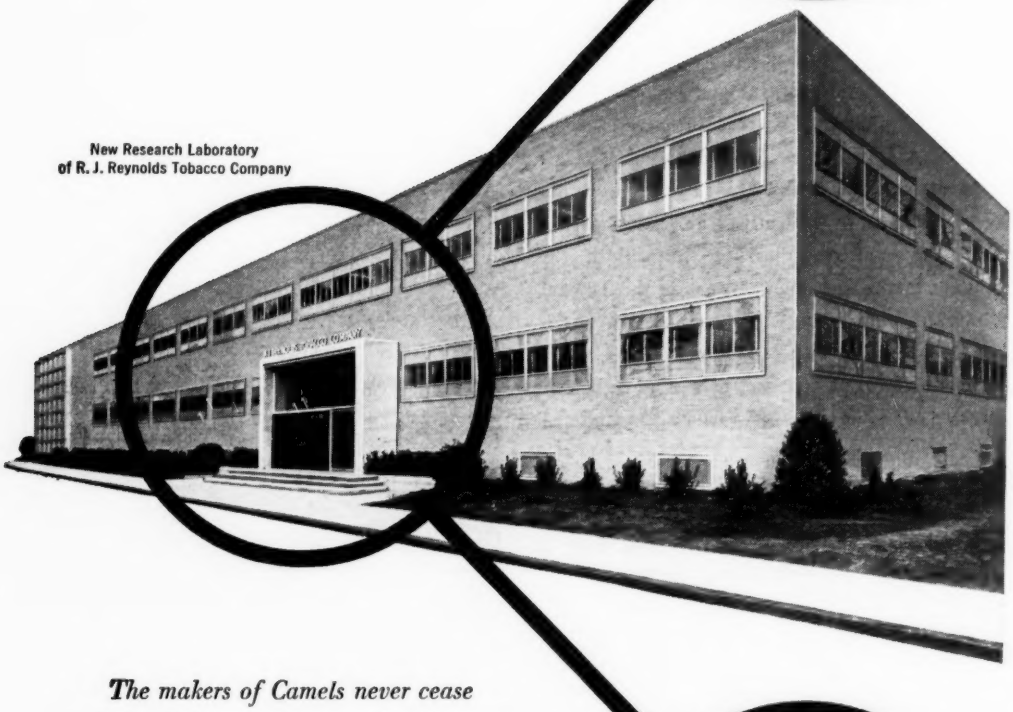
PROGRESS THROUGH RESEARCH



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SCHEDULE OF MEETINGS

ORGANIZATION	PRESIDENT	SECRETARY	ANNUAL MEETING
Florida Medical Association	Frederick K. Herpel, W. Palm Bch.	Samuel M. Day, Jacksonville	Hollywood, Apr. 25-28, '54
Florida Medical Districts	John D. Milton, Miami	Council Chairman	
A-Northwest	Francis M. Watson, Marianna	George S. Palmer, Tallahassee	Tallahassee, Oct. 19, '53
B-Northeast	William C. Thomas, Jr., Gainesville	Thomas C. Kenaston, Cocoa	St. Augustine, Oct. 20, '53
C-Southwest	Emmett E. Martin, Haines City	Clyde O. Anderson, St. Petersburg	Tampa, Oct. 21, '53
D-Southeast	Erasmus B. Hardee, Vero Beach	Russell B. Carson, Ft. Lauderdale	West Palm Beach, Oct. 22, '53
Florida Specialty Societies			
Academy of General Practice	Raymond R. Killinger, Jacksonville	Leo M. Wachtel, Jr., Jacksonville	Hollywood, Apr. 25, '54
Allergy Society	James H. Putman, Miami	Lewis Palay, Miami Beach	" "
Anesthesiologists, Soc. of	Adelbert F. Schrimer, Orlando	Breckenridge W. Wing, Orlando	" "
Chapter, Am. Coll. Chest Phys.	Nathaniel M. Levin, Miami	Hawley H. Seiler, Tampa	" "
Derm. and Syph., Soc. of	Morris Waisman, Tampa	Joseph A. J. Farrington, Jacksonville	" "
Health Officers' Society	Lorenzo L. Parks, Jacksonville	Clarence L. Brumback, W. Palm Beach	" "
Industrial & Railway Surgeons	Lloyd J. Netto, West Palm Beach	John H. Mitchell, Jacksonville	" "
Neurology & Psychiatry	William H. McCullagh, Jacksonville	Roger E. Phillips, Orlando	" "
Ob. and Gynec. Society	Ferdinand Richards, Jacksonville	J. Champneys Taylor, Jacksonville	" "
Ophthal. & Otol., Soc. of	Mozart A. Lischkoff, Pensacola	Carl S. McLemore, Orlando	" "
Orthopedic Society	Herschel G. Cole, Tampa	Newton C. McCollough, Orlando	Tampa, Oct., '53
Pathologists, Society of	Alfred E. Cronkite, Fort Lauderdale	Clarence W. Ketchum, Tallahassee	Hollywood, Apr. 25, '54
Pediatric Society	Charlotte C. Maguire, Orlando	Wesley S. Nock, Coral Gables	
Proctologic Society	John J. Cheleden, Daytona Beach	George Williams, Jr., Miami	Hollywood, Apr. 25, '54
Radiological Society	Nelson T. Pearson, Miami	Hugh G. Reaves, Sarasota	" "
Urological Society	Frank M. Woods, Miami	David W. Goddard, Daytona Beach	" "
Florida—			
Basic Science Exam. Board	Mr. Paul A. Vestal, Winter Park	M. W. Emmel, D.V.M., Gainesville	Gainesville, Nov. 7, '53
Blood Banks, Association	James N. Patterson, Tampa	Sherman B. Forbes, Tampa	
Blue Cross of Florida, Inc.	Mr. C. Dewitt Miller, Orlando	Mr. H. A. Schroder, Jacksonville	Miami, Dec. 3, '53
Blue Shield of Florida, Inc.	Leigh F. Robinson, Ft. Lauderdale	Webster Merritt, Jacksonville	Jacksonville, May '54
Cancer Council	Ashbel C. Williams, Jacksonville	Lorenzo L. Parks, Jacksonville	Hollywood, Apr. 25, '54
Clinical Diabetes Assn.	Fred Mathers, Orlando	Edward R. Smith, Jacksonville	Orlando, 1954
Dental Society, State	L. M. Schulstad, D.D.S., Bradenton	B. S. Carroll, D.D.S., Jacksonville	Daytona Beach, April '54
Heart Association	Jere W. Annis, Lakeland	William P. Hixon, Pensacola	
Hospital Association	Mr. T. F. Little, Daytona Beach	Mr. Tracy B. Hare, Miami	Miami, Nov. '53
Medical Examining Board	Ralph B. Spires, DeFuniak Springs	Homer L. Pearson, Jr., Miami	
Medical Postgraduate Course	Turner Z. Cason, Jacksonville	Chairman	
Nurses Association, State	Mrs. Mary Livingston, W. P. Beach	Mrs. Idalyn Lawthon, Tampa	Tampa, Nov. '53
Pharmaceutical Association, State	Mr. A. W. Morrison, Miami	Mr. R. Q. Richards, Ft. Myers	Jacksonville, May '54
Public Health Association	Mr. Angus Laird, Tallahassee	Mr. Fred B. Ragland, Jacksonville	Tampa, Oct. 22-24, '53
Trudeau Society	Hawley H. Seiler, Tampa	John G. Chesney, Miami	
Tuberculosis & Health Assn.	Leffie M. Carlton, Jr., Tampa	Mrs. L. C. Conant, Fort Myers	
Woman's Auxiliary	Mrs. Thomas C. Kenaston, Cocoa	Mrs. Lee Rogers, Jr., Cocoa	Hollywood, Apr. 25-27, '54
American Medical Association	Edward J. McCormick, Toledo, O.	Geo. F. Lull, Chicago	San Francisco, June 21-25, '54
A.M.A. Clinical Session	Edward J. McCormick, Toledo, O.	Geo. F. Lull, Chicago	St. Louis, Dec. 1-4, '53
Southern Medical Association	Walter C. Jones, Miami	Mr. C. P. Loranz, Birmingham	Atlanta, Oct. 26-29, '53
Alabama Medical Association	D. O. Morgan, Gadsden	Douglas L. Cannon, Montgomery	Mobile, Apr. 15-17, '54
Georgia, Medical Assn. of	Wm. P. Harbin, Jr., Rome	David Henry Poer, Atlanta	Macon, May 2-5, '54
S. E. Hospital Conference	Charles W. Holmes, Memphis, Tenn.	Pat Groner, Pensacola	Atlanta, Apr. 6-8, '54
Southeastern Allergy Assn.	Walker L. Rucks, Memphis, Tenn.	Kath. B. MacInnis, Columbia, S. C.	Atlanta, '54
Southeastern, Am. Urological Assn.	Russell B. Carson, Fort Lauderdale	Sidney Smith, Raleigh, N. C.	Palm Beach, April, '54
Southeastern Surgical Congress	J. R. Young, Anderson, S. C.	B. T. Beasley, Atlanta	Birmingham, Mar. 8-11, '54
Gulf Coast Clinical Society	Hays Zieman, Mobile, Ala.	William Atkinson, Mobile, Ala	Mobile, Oct., '53

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	SOCIETY	PRESIDENT	SECRETARY	MEETING DATE	MEMBERS TOTAL	MEMBERS PAID	COUNCILOR
A	Bay	Harold E. Wager, M.D. Box 984 Panama City	Charles H. Daffin, M.D. 536 E. 4th St. Panama City	1st Tuesday 7:30 P.M.	24	22	
	Escambia *Santa Rosa	Alvin L. Stebbins, M.D. 621 N. O St. Pensacola	Paul F. Baranco, M.D. 608 Blount Bldg. Pensacola	2nd Tuesday 8:00 P.M.	91	100%	
	Franklin-Gulf	Photis J. Nichols, M.D. Apalachicola	Terry Bird, M.D. Apalachicola	Last Wednesday except Dec. & July	7	100%	A-1-54 Francis M. Watson, M.D. Marianna
	Jackson-Calhoun	Grayson C. Snyder, M.D. Floyd Bldg. Blountstown	Francis M. Watson, M.D. 120 Deering St. Marianna	1st Thursday 7:00 P.M. March, June, Sept., Dec.	18	100%	
	Walton-Okaloosa	Edgar H. Myers, M.D. DeFuniak Springs	William D. Cawthon, M.D. DeFuniak Springs	3rd Thursday 8:00 P.M.	17	100%	
	Washington-Holmes	L. H. Paul, M.D. Bonifay	Bayllye W. Dalton, M.D. Chipley		5	100%	
	Columbia *Baker, Hamilton	Robert B. Harkness, M.D. 504 E. Duval St. Lake City	Thomas H. Bates, M.D. 27 W. Madison St. Lake City	1st Monday 7:30 P.M.	14	10	
	Leon-Gadsden-Liberty-Wakulla-Jefferson	Charles F. James, Jr., M.D. Washington Sq. Bldg. Tallahassee	T. Bert Fletcher, Jr., M.D. 516 N. Adams St. Tallahassee	Quarterly 7:30 P.M.	60	55	
	Suwannee	Hiram B. Curry, M.D. Jasper	Edward G. Haskell, Jr., M.D. Branford	1st Saturday 9:30 A.M.	8	100%	A-2-55 George S. Palmer, M.D., Tallahassee
	Madison	Julian M. DuRant, M.D. Madison	A. Franklin Harrison, M.D. Madison		4	3	
	Taylor *Dixie, Lafayette	Mark E. Adams, M.D. Perry	Walter J. Baker, M.D. Foley	Last Friday 8:00 P.M.	4	100%	252
B	Alachua *Bradford, Gilchrist, Union	F. Emory Bell, M.D. Box 113 Gainesville	Winston L. Summerlin, M.D. 111 S. W. 3rd St. Gainesville	2nd Tuesday 8:00 P.M.	44	42	
	Duval *Clay	W. W. Rogers, M.D. 962 Main St. Jacksonville	John T. Stage, M.D. 2033 Riverside Ave. Jacksonville	1st Tuesday 8:15 P.M.	258	228	
	Marion *Levy	Eugene G. Peek, Jr., M.D. Box 248 Ocala	Bertrand F. Drake, M.D. Box 326 Dunnellon	3rd Tuesday 7:00 P.M.	27	100%	B-3-54 William C. Thomas, Jr., M.D. Gainesville
	Nassau	David G. Humphreys, M.D. 113 N. 6th St. Fernandina	John W. McClane, M.D. Fernandina	Last Friday 8:00 P.M.	12	100%	
	Putnam	Bernard E. Kane, M.D. Box 216 Crescent City	James A. Long, Jr., M.D. 1001 Main St. Palatka	2nd Tuesday 6:00 P.M.	9	100%	
	St. Johns	John M. Canakaris, M.D. Box 295 Bunnell	James J. DeVito, M.D. Box 100 St. Augustine	3rd Tuesday 8:30 P.M.	15	100%	
C	Brevard	Laurent L. LaRoche, M.D. 526 Brevard Ave. Cocoa	Allen E. Kuester, M.D. 501 Delannoy Ave. Cocoa	2nd Tuesday	25	24	
	Lake *Sumter	John D. Bloom, M.D. Groveland	J. Basil Hall, M.D. Tavares	1st Wednesday 7:30 P.M.	29	28	
	Orange *Osceola	G. Tayloe Gwathmey, M.D. 417 E. Robinson Ave. Orlando	Andrew W. Townes, Jr., M.D. 717 N. Magnolia Ave. Orlando	3rd Wednesday 8:00 P.M.	172	169	B-4-55 Thomas C. Kenaston, M.D., Cocoa
	Seminole	Wade H. Garner, M.D. Box 219 Sanford	Frank L. Quillman, M.D. Box 158 Sanford	2nd Tuesday 5:30 P.M.	18	100%	
	Volusia *Flagler	Morris B. Seltzer, M.D. 614 N. Peninsula Dr. Daytona Beach	Robert L. Miller, M.D. 258½ S. Beach St. Daytona Beach	2nd Tuesday 7:30 P.M.	83	75	692
	Hillsborough	Arthur R. Knauf, M.D. 706 Franklin St. Tampa	Julien C. Pate, Jr., M.D. 1105 First Nat. Bk. Bldg. Tampa	1st Tuesday 8:00 P.M.	176	169	
	Manatee	William D. Sugg, M.D. Bradenton Bank Bldg. Bradenton	Marjorie L. Warner, M.D. 202 Manatee Ave., E. Bradenton	2nd Tuesday 8:30 P.M.	21	20	
	Pasco-Hernando-Citrus	Karl T. Humes, M.D. Box 157 Bushnell	W. Wardlaw Jones, M.D. Box 247 Dade City	2nd Thursday 7:00 P.M.	18	100%	C-5-55 Clyde O. Anderson, M.D. St. Petersburg
	Pinellas	Everett M. Harrison, M.D. 1601 N. Ft. Harrison Ave. Clearwater	Whitman C. McConnell, M.D. 1517 4th St., N. St. Petersburg	1st Mon. Even Mo. 1st Fri. Odd Mo. 6:00 P.M.	205	202	
	Sarasota	Cecil E. Miller, M.D. 880 S. Orange Ave. Sarasota	Melvin M. Simmons, M.D. 816 Florasota Gardens Sarasota	2nd Tuesday 8:30 P.M.	47	44	
D	DeSoto-Hardee-Highlands-Glades	Wesley S. Pyatt, M.D. Bowling Green	Merle C. Kayton, M.D. Wauchula	2nd Tuesday 8:00 P.M.	24	100%	
	Lee-Charlotte-Collier-Hendry	Ernest Bostelman, M.D. 201 Pythian Bldg. Fort Myers	Harry Fagan, Jr., M.D. 310 Richards Bldg. Fort Myers	3rd Monday 7:30 P.M.	29	100%	C-6-54 Emmett E. Martin, M.D. Haines City
	Polk	Jere W. Annis, M.D. Box 1021 Lakeland	James T. Shelden, M.D. Box 1021 Lakeland	2nd Wednesday 7:00 P.M.	95	94	615
	Indian River	John P. Gifford, M.D. 1625 Osceola Blvd. Vero Beach	William L. Fitts, 3rd, M.D. Vero Beach Arcade Vero Beach	2nd Tuesday 8:00 P.M.	8	100%	
	Palm Beach	Graham W. King, Jr., M.D. Box 505 Delray Beach	David A. Newman, M.D. 175 Bradley Pl. Palm Beach	3rd Monday 8:00 P.M.	123	100%	D-7-54 Erasmus B. Hardee, M.D. Vero Beach
	St. Lucie-Okeechobee-Martin	John T. McDermid, M.D. 337 N. 4th St. Fort Pierce	Adrian M. Sample, M.D. Box 897 Fort Pierce	3rd Thursday 8:00 P.M.	17	16	
	Broward	Claus A. Peterson, M.D. 312 Blount Bldg. Ft. Lauderdale	James M. Weaver, M.D. Box 4084 Ft. Lauderdale	4th Tuesday 8:00 P.M.	96	95	
	Dade	Ralph W. Jack, M.D. 1700 Biscayne Blvd. Miami	Walter W. Sackett, Jr., M.D. 2500 Coral Way Miami	1st Tuesday 8:30 P.M.	631	566	D-8-55 Russell B. Carson, M.D. Fort Lauderdale
	Monroe	Joseph L. G. Lester, Jr., M.D. 422 Fleming St. Key West	Allen S. Shepard, M.D. 638 United St. Key West	2nd Thursday 8:00 P.M.	15	13	890

*Supervise and aid until organized separately.

Total 2,449